



## ESSENTIALS OF GENERAL ANÆSTHESIA



# *Essentials of* GENERAL ANÆSTHESIA

By R. R. MACINTOSH,  
M.A., M.D., F.R.C.S. (Ed.)

*Hon. M.D. Honorary Assistant Professor of Anaesthetics  
University of Oxford. Consultant in Anaesthetics  
at the Royal Air Force Anaesthetist, United Oxford Hospitals,  
Fellow for the D.A. Fellow of the Faculty of Anaesthetists  
Royal College of Surgeons*

and  
IRIDA B. BANNISTER,  
M.A., M.D., D.A.

*Consultant Anaesthetist, Wolverhampton Group of Hospitals  
Birmingham Regional Hospital Board. Fellow of the Faculty  
of Anaesthetists, Royal College of Surgeons. Late First Assistant  
Newfield Department of Anaesthetics, University of Oxford  
Temp. Major R.A.M.C.*

*Illustrated by*  
MISS M. C. McILROY

FIFTH EDITION

BLACKWELL  
SCIENTIFIC PUBLICATIONS  
OXFORD



*This book is copyright. It may not be reproduced by any means in whole or in part without permission. Application with regard to copyright should be addressed to the publishers.*

*Published simultaneously in the United States of America by Charles C Thomas, Publisher, 301-327 East Lawrence Avenue, Springfield, Illinois.*

*Published simultaneously in Canada by The Ryerson Press, 299 Queen Street West, Toronto 2.*

FIRST PRINTED	1940
SECOND EDITION	1941
THIRD EDITION	1943
THIRD EDITION	REPRINTED 1943
THIRD EDITION	REPRINTED 1945
FOURTH EDITION	1947
FIFTH EDITION	1952

## PREFACE

THIS book is based on a series of lectures given to students at the Dental School of University College Hospital London, and the University of Oxford Medical School. In presenting it we hope that it will assist not only those seeking an introduction to anaesthetics or preparing for their final examinations but also the medical or dental practitioner who desires to refresh his knowledge of the essentials of general anaesthesia.

The lack of a text book suitable as a supplement to undergraduate lectures and the practical lessons learned in the operating room has induced us to attempt a statement of the foundation of principles underlying the administration of anaesthetics in general. The case histories cited however in the main have been chosen deliberately from the field of dental surgery since the art of dental anaesthesia is often much neglected and in our opinion a mastery of the difficulties encountered there will help the anaesthetist to understand and overcome many of the problems he is likely to meet in anaesthesia for operations on other parts of the body. In order to clarify the descriptions given in the text we have made full use of illustrations.

The standard of administration of anaesthetics by the family doctor is a standing joke between dentists and is frequently a matter of comment among general surgeons. Although the criticism is amply justified the reproach itself is unfair inasmuch as a general practitioner has little more opportunity of reaching the standard attained by the specialist in anaesthetics than he has of becoming a specialist in any other branch of medicine or surgery. It must be admitted, however, that the average doctor while fully recognising that he has something to learn in other fields is apt to be unduly complacent about his knowledge of anaesthesia and the technique of its administration especially for dental operations.

At many general hospitals extractions are performed only in cases of emergency non urgent work is referred to dental hospitals. The doctor's early experience in anaesthesia for dental work is often therefore confined to the administration of nitrous oxide in cases where an acute alveolar abscess necessitates extraction of a tooth. Such an extraction is usually easy and a short and not necessarily skilful anaesthesia frequently suffices. The average medical student has, in

fact little opportunity of seeing and far less of practising the refinements of anæsthesia required for operations in the mouth. We have therefore incorporated in this book practical hints which should prove useful to the general practitioner with limited experience of this subject, and even to the anæsthetist who devotes but little of his time to this branch. Gone are the days when it was considered sufficient for the doctor to present the dentist with an unconscious patient and then to walk round the room leaving the supervision of the patient and the extraction of the teeth to the dentist. The production and maintenance of surgical anæsthesia should be regarded both as an art and as a science in dental work particularly the former plays an important role and the anæsthetist must realise that it is his duty to co-operate with the dentist in every possible way.

The anæsthetist must train himself to recognise at a glance the type temperament and physical condition of the patient. He must know the significance of breathlessness of œdema of protruding eyes of plethora and of anæmia and he must recognise the signs of alcoholism. He will soon learn that pallor does not necessarily indicate anæmia nor a rapid pulse cardiac disorder. He must interpret correctly the cause of any cyanosis which may develop during anæsthesia and be technically expert enough to deal with all the incidents whether grave or trifling which may occur during narcosis. He must be constantly alert to observe every indication however small of a change in the patient's reactions to the anæsthetic and he must be alive to the importance of maintaining a clear airway. He has the entire responsibility for the patient's safety during unconsciousness and thus for his life and this responsibility is no whit less serious during anæsthesia for dental extractions than during anæsthesia for more severe operations. This is often inadequately appreciated, since the patient is rarely ill and the operation generally lasts only a short time.

The anæsthetist has always three people to satisfy—the patient the surgeon and himself. Although it is to be hoped that he will never succeed in satisfying himself completely he can succeed in safeguarding his patient and if he is fortunate in satisfying the surgeon or dentist with whom he co-operates.

We are greatly indebted to Miss Marjorie R. Gibson for her invaluable care and patience in dealing with our manuscripts and to Miss M. C. McLarty for her skill and attention to detail in the preparation of the majority of the illustrations. Our thanks are also due to Miss A. J. Arnott for the remainder of the drawings to Miss M.

Herring Shaw for the photograph, and to Mr. R. M. Duncan, D.Phil. for assistance with the chapter on histology.

As anaesthetists frequently refer to nitrogen, oxide, carbon dioxide and oxygen by their chemical symbols, we have used these abbreviations ( $N_2O$ ,  $CO_2$  and  $O_2$ ) where we might have done so in conversation or in making a case record.

OXFORD

*November 1941*

## PREFACE TO FIFTH EDITION

The changes in this edition have been effected by the undersigned. They include a chapter on Curare and other muscle relaxants which have been introduced into anaesthesia during the last few years, and additions and modifications in other chapters which the advent of these drugs has made necessary. There are also various other small additions and changes.

IRIDA B. BANNISTER  
(Mrs. R. U. Whitney)

BROOKLYN, NEW YORK  
*June 1951*

fact little opportunity of seeing and far less of practising the refinements of anæsthesia required for operations in the mouth. We have therefore incorporated in this book practical hints which should prove useful to the general practitioner with limited experience of this subject and even to the anæsthetist who devotes but little of his time to this branch. Gone are the days when it was considered sufficient for the doctor to present the dentist with an unconscious patient and then to walk round the room leaving the supervision of the patient and the extraction of the teeth to the dentist. The production and maintenance of surgical anæsthesia should be regarded both as an art and as a science. In dental work particularly the former plays an important role and the anæsthetist must realise that it is his duty to co-operate with the dentist in every possible way.

The anæsthetist must train himself to recognise at a glance the type temperament and physical condition of the patient. He must know the significance of breathlessness, of œdema of protruding eyes of plethora and of anæmia and he must recognise the signs of alcoholism. He will soon learn that pallor does not necessarily indicate anæmia nor a rapid pulse cardiac disorder. He must interpret correctly the cause of any cyanosis which may develop during anæsthesia and be technically expert enough to deal with all the incidents whether grave or trifling which may occur during narcosis. He must be constantly alert to observe every indication however small of a change in the patient's reactions to the anæsthetic and he must be alive to the importance of maintaining a clear airway. He has the entire responsibility for the patient's safety during unconsciousness and thus for his life and this responsibility is no whit less serious during anæsthesia for dental extractions than during anæsthesia for more severe operations. This is often inadequately appreciated since the patient is rarely ill and the operation generally lasts only a short time.

The anæsthetist has always three people to satisfy—the patient, the surgeon and himself. Although it is to be hoped that he will never succeed in satisfying himself completely, he can succeed in safe-guarding his patient and if he is fortunate in satisfying the surgeon or dentist with whom he co-operates.

We are greatly indebted to Miss Marjorie R. Gibson for her invaluable care and patience in dealing with our manuscripts and to Miss M. C. McLarty for her skill and attention to detail in the preparation of the majority of the illustrations. Our thanks are also due to Miss A. J. Arnott for the remainder of the drawings to Miss M.

# CONTENTS

PREFACE	v
PREFACE TO THIRD EDITION	vii
CONTENTS	
I HISTORY OF ANAESTHESIA	1
II CENTRAL ANAESTHESIA	13
III THEORY OF ANAESTHESIA	19
IV INSPIRATION	29
V CYANIDS	51
VI SIGNS OF ANAESTHESIA	62
VII SIGNS OF NITROUS-OXIDE ANAESTHESIA	68
VIII INDICATIONS FOR LOCAL OR CENTRAL ANAESTHESIA IN DENTISTRY	75
IX CHOICE OF CENTRAL ANAESTHETIC	79
X TRI ANAESTHETIC MEDICATION	88
FOR THE AMBULATORY PATIENT	93
FOR THE HOSPITALISED PATIENT	95
MORPHIA	95
HYOSCINI (SCOPOLAMINI)	96
ALCOHOL	97
BASAL ANAESTHESIA	99
THE RECTAL ROUTE	101
AVERTIN	102
PARALDEHYDE	102
THIOFENTONE AND HEXOBARBITONE	103
THE ORAL ROUTE	103
NEMBUTAL	104
THE INTRAVENOUS ROUTE	104
THIOFENTONE AND HEXOBARBITONE	104
NEMBUTAL	105
ATROPINE	105
XI THE MORE DIFFICULT PATIENT	107



I	DOTPACHIAL ANÆSTHESIA	246
	MECHANICS OF LARYNGOSCOPY	247
	DIRECT METHOD OF LARYNGOSCOPY	249
	DIFFICULTY IN INTUBATING THE CORDS	257
	BLIND NASAL INTUBATION	260
	INTUBATION UNDER LOCAL ANÆSTHESIA	265
	WARNING OF THE END OF DOTPACHIAL ANÆSTHESIA	266
	LARYNGOSCOPIES	267
VI	SCOUTING ACTION OF TACKS AND TONGUE FORCES	271
VII	CARE OF THE UNCONSCIOUS PATIENT	285
III	PHARYNGEAL OBSTRUCTION	291
	TACKWARD DISPLACEMENT OF THE TONGUE	291
	FOREIGN BODIES	297
	LARYNGEAL SPASM	298
	CEDEMA OF THE CLOTIS	301
XIX	EMERGENCIES	305
	FOREIGN BODIES LOST IN THE MOUTH	306
	FOREIGN BODIES IN THE ALIMENTARY TRACT	307
	FOREIGN BODIES IN THE AIR PASSAGES	308
	ACUTE RESPIRATORY OBSTRUCTION	309
	LARYNGEAL SPASM	310
	IMMERSION OF A FOREIGN BODY	313
	EMERGENCY TRACHEOTOMY	315
	LARYNGOTOMY	325
	OVERDOSE OF ANÆSTHETIC	327
	ARTIFICIAL RESPIRATION	327
	CARDIAC FAILURE	332
XXX	CYLINDRICAL VALVES AND REDUCING VALVES	334
XXVI	ANALGESIA	339
XXXII	THE OXFORD VALORISER	347
XXXIII	LIGAL	356
	INDEX OF PERSONAL NAMES	366
	INDEX	369



## CONTENTS

### CH PT R

- XII CHILDREN
- XIII STATUS LYMPHATICUS
- XIV PREPARATION FOR OPERATION
- XV BARBITURATES
- XVI ETHYL CHLORIDE
- XVII VINESTHENE
- XVIII TRICHLORETHYLENE (TRILENE)
- XIX ETHER
- XX CHLOROFORM
- XXI CYCLOPROPANE
- XXII CURARE AND OTHER MUSCLE RELAXANTS
  - PHARMACOLOGY AND PHYSIOLOGY OF *D* TUBOCURARINE  
AND ITS ANTIDOTE PROSTIGMIN
  - USE OF *D* TUBOCURARINE CHLORIDE IN ANÆSTHESIA
  - OTHER MUSCLE RELAXANTS
    - FLAXIDIL
    - C 10
    - MYANESIN
- XXIII PRELIMINARY APPROACH TO DENTAL ANÆSTHESIA
- XXIV NITROUS OXIDE
  - PHYSICAL PROPERTIES
  - GENERAL CONSIDERATIONS
  - APPARATUS FOR USE IN THE DENTAL SURGERY
  - COMMON ERRORS IN ADMINISTRATION
  - PREVENTION OF DISCOMFORT DURING INDUCTION
  - INDUCTION
  - ESTABLISHMENT OF NASAL RESPIRATION
  - MAINTENANCE OF ANÆSTHESIA
  - POSITIONING THE HEAD TO ALLOW ACCESS TO SPECIAL  
TEETH
  - RECOVERY

XXV	THE LARYNX	25
	ANATOMY OF THE LARYNX	26
	PHYSIOLOGY OF THE LARYNX	27
	THE LARYNX IN DYSPHAGIA	28
	THE LARYNX IN THE LARYNGEAL CLEFT	29
	THE LARYNX IN THE LARYNGEAL CLEFT	30
XXVI	THE LARYNX	31
XXVII	THE LARYNX	32
XXVIII	THE LARYNX	33
	THE LARYNX	34
	THE LARYNX	35
	THE LARYNX	36
	THE LARYNX	37
	THE LARYNX	38
	THE LARYNX	39
	THE LARYNX	40
	THE LARYNX	41
	THE LARYNX	42
	THE LARYNX	43
	THE LARYNX	44
	THE LARYNX	45
	THE LARYNX	46
	THE LARYNX	47
	THE LARYNX	48
	THE LARYNX	49
	THE LARYNX	50
	THE LARYNX	51
	THE LARYNX	52
	THE LARYNX	53
	THE LARYNX	54
	THE LARYNX	55
	THE LARYNX	56
	THE LARYNX	57
	THE LARYNX	58
	THE LARYNX	59
	THE LARYNX	60
	THE LARYNX	61
	THE LARYNX	62
	THE LARYNX	63
	THE LARYNX	64
	THE LARYNX	65
	THE LARYNX	66
	THE LARYNX	67
	THE LARYNX	68
	THE LARYNX	69
	THE LARYNX	70
	THE LARYNX	71
	THE LARYNX	72
	THE LARYNX	73
	THE LARYNX	74
	THE LARYNX	75
	THE LARYNX	76
	THE LARYNX	77
	THE LARYNX	78
	THE LARYNX	79
	THE LARYNX	80
	THE LARYNX	81
	THE LARYNX	82
	THE LARYNX	83
	THE LARYNX	84
	THE LARYNX	85
	THE LARYNX	86
	THE LARYNX	87
	THE LARYNX	88
	THE LARYNX	89
	THE LARYNX	90
	THE LARYNX	91
	THE LARYNX	92
	THE LARYNX	93
	THE LARYNX	94
	THE LARYNX	95
	THE LARYNX	96
	THE LARYNX	97
	THE LARYNX	98
	THE LARYNX	99
	THE LARYNX	100



# CHAPTER I

## HISTORY OF ANAESTHESIA

MAN'S attempts to produce insensibility to pain are of very long standing. The Assyrians were aware of the anodyne and soporific properties of poppy and mandrake<sup>1</sup> and we know from the Homeric reference to nepenthes<sup>2</sup> that the Greeks alleviated mental and probably physical suffering by narcotic drinks. Theophrastus (370-286 B.C.) the protobotanist specifically tells us that the root of mandrake steeped in wine produces sleep<sup>3</sup>.

The Romans also used decoctions of mandrake in alcohol. Pliny (A.D. 23-79) says of the juice of the mandrake. Administered in doses proportional to the strength of the patient this juice has a narcotic effect. It is given for injuries inflicted by serpents and before incisions or punctures are made in the body in order to ensure insensibility to the pain and he adds, Indeed, for this last purpose with some persons the odour of it is quite sufficient to induce sleep<sup>4</sup>. Dioscorides also who was a Greek surgeon in the army of Nero from A.D. 54 to 68 and compiled the first great work on materia medica prescribed mandrake for the relief of pain, and stated that according to the preparation it might either be drunk or inhaled, or given in an enemata<sup>5</sup>.

Analgesic potions in fact have been well known for more than two thousand years but although described in medical text books throughout the mediæval and early Renaissance periods and sporadically even until the end of the eighteenth century their utilisation seems to have been the exception rather than the rule. Although on empirical grounds early physicians recognised that certain plants possessed narcotic properties they had no means of assessing the potency of the extracts employed. Different samples of the same herb differ considerably in the amounts of principle contained. Thus, though a measured dose of one might produce the required depth of unconsciousness another sample similarly prepared might be depression,

and disaster  
hazardous



## CHAPTER I

# HISTORY OF ANAESTHESIA

MAN'S attempts to produce insensibility to pain are of very long standing. The Assyrians were aware of the anodyne and soporific properties of poppy and mandrake<sup>1</sup> and we know from the Homeric reference to nepenthes<sup>2</sup> that the Greeks alleviated mental and probably physical suffering by narcotic drinks. Theophrastus (370-286 B.C.) the protobotanist specifically tells us that the root of mandrake steeped in wine produces sleep<sup>3</sup>.

The Romans also used decoctions of mandrake in alcohol. Pliny (A.D. 23-79) says of the juice of the mandrake, Administered in doses proportional to the strength of the patient, this juice has a narcotic effect. It is given for injuries inflicted by serpents and before incisions or punctures are made in the body in order to ensure insensibility to the pain and he adds. Indeed, for this last purpose with some persons the odour of it is quite sufficient to induce sleep<sup>4</sup>. Dioscorides also who was a Greek surgeon in the army of Nero from A.D. 54 to 68 and compiled the first great work on materia medica prescribed mandrake for the relief of pain, and stated that according to the preparation it might either be drunk, or inhaled, or given in an enema<sup>5</sup>.

Analgesic potions in fact have been well known for more than two thousand years but although described in medical text-books throughout the medieval and early Renaissance periods and sporadically even until the end of the eighteenth century their utilisation seems to have been the exception rather than the rule. Although on empirical grounds early physicians recognised that certain plants possessed narcotic properties they had no means of assessing the potency of the extracts employed. Different samples of the same herb differ considerably in the amounts of active principle contained. Thus though a measured dose of one extract of poppy might produce the required depth of unconsciousness the same dose of another sample similarly prepared might be either entirely inadequate or might produce extreme depression or even death. The prudent surgeon therefore while acknowledging the successes was deterred by the numerous failures and disasters from using so uncertain a means of preventing pain. So hazardous was the method considered that until the middle of the

nineteenth century an unfortunate patient about to be operated upon found himself bound and held down by strong men his only help being fortitude or unconsciousness from fainting <sup>6</sup>

During the seventeenth and eighteenth centuries opium had become increasingly important as an anodyne and narcotic as indeed the word *opiate* itself suggests. It was sometimes given in an enema as a mild sedative before operations but more frequently after the operation. In extremely painful conditions held to be inoperable where pain could not be relieved otherwise its use was and still is, considered essential for mitigating the sufferings of the patient's last days. It was universally recognised that opium like alcohol if pushed to excess produced complete insensibility but this use of these drugs was vigorously condemned by physicians and surgeons of good repute as a dangerous and unjustified practice <sup>7</sup>

Administration of narcotics in known doses first became possible in 1806 when Serturner <sup>8</sup> succeeded in isolating morphine the chief alkaloid of opium. Its routine use in medicine in place of opium dates from about 1820 and was due to the French physiologist Magendie <sup>9</sup> (1783-1855) who fully appreciated that the superiority of the pure alkaloids lay in the fact that they could be given in known doses.

In adopting morphine Magendie prescribed it only by mouth. Though the isolation of this alkaloid made it possible for physicians to administer a dose of constant composition the results continued to be inconsistent because of the variations in the rate of absorption of drugs given by mouth (p. 103). It was not until five years after the discovery of ether that the hypodermic syringe was introduced by Pravaz in 1851 and independently by Alexander Wood in 1853. By administering morphine hypodermically the rate of absorption was brought under control but since inhalation anæsthesia had already been enthusiastically adopted there was now no incentive to exploit to the full the possibility of producing narcosis with this drug.

The work of Priestley (1733-1804) and Lavoisier (1743-1794) on oxygen and the nature of respiration turned medical thought in a new direction and led to a theory that some diseases might be benefited by inhaling oxygen or other gases. In 1796 Thomas Beddoes founded at Bristol a Medical Pneumatic Institution where experiments and treatment along such lines could be carried out <sup>10</sup>. He commissioned James Watt to design the apparatus required and appointed Humphry Davy (1778-1829) as superintendent of the Institution.

During his investigations into the properties of nitrous oxide Davy in 1799 verified by animal experiment that the gas was respirable he then inhaled it himself and stumbled upon its analgesic properties. In

1800 he published an exhaustive monograph<sup>11</sup> in a comparatively small section of which he records his mental and physiological reactions on inhaling the gas and states that inspiration was accompanied with loss of distinct sensation and voluntary power, and that it would relieve headache. On p. 464 he writes that the power of the immediate operation of the gas in removing intense physical pain I had a very good opportunity of ascertaining. In cutting one of the unlucky teeth called *dentes sapientiae* I experienced an extensive inflammation of the gum accompanied with great pain. On the day when the inflammation was most troublesome I breathed three large doses of nitrous oxide. The pain always diminished after the first four or five inspirations. As the former state of mind however returned the state of organ returned with it and once I imagined that the pain was more severe after the experiment than before.

Davy's somewhat lengthy summary of his conclusions includes the brief statement. As nitrous oxide in its extensive operation appears capable of destroying physical pain it may probably be used with advantage during surgical operations in which no great effusion of blood takes place. These few words lifted from the unimportant position assigned to them by Davy among a mass of his other deductions not all of them accurate have been given much prominence in the history of anaesthesia and it is often implied that Davy was suggesting that nitrous oxide should be used as a general anaesthetic. It is probable however that no such idea occurred to him, even though he knew that prolonged inhalation of the gas would produce stupor. If indeed he had conceived the idea of producing surgical anaesthesia by means of nitrous oxide he would deserve blame rather than praise in that he fresh from apprenticeship to a surgeon made no attempt either to follow up this important idea himself or to encourage others to do so. It should however be realised that Davy was then only twenty two years old and that his dominant enthusiasm was chemistry rather than medicine as was shown by the fact that a year later he resigned his post at the Pneumatic Institution in order to take charge of the chemical laboratory of the newly founded Royal Institution in London. This early promotion resulted in the discontinuance of Davy's experiments with nitrous oxide. Had he remained at Bristol and taken his medical degree as originally intended it is more than possible that his genius would have led him to utilise nitrous oxide as a means of producing surgical anaesthesia.

At about the time that Davy's interest in nitrous oxide began to wane Henry Hill Hickman (1800-1830) was born. In 1820 he was



admitted a member of the Royal College of Surgeons, and set up in practice in Shropshire. Although little older than Davy had been when he carried out the researches on nitrous oxide. Hickman differed from Davy in that he had the clearly defined object of showing that surgical anæsthesia could be achieved by inhalation. He formulated this belief in a pamphlet published in 1824.<sup>1</sup> In a letter addressed to his friend T. A. Knight F.R.S. he says: "There is not an individual who does not shudder at the idea of an operation" and I have frequently lamented when performing my own duties as a Surgeon that something has not been thought of whereby the fears may be tranquilised and suffering relieved. Above all from the many experiments on suspended Animation I have wondered that some hint has not been thrown out of its probable utility and noticed by Surgeons and consequently I have been induced to make experiments on Animals endeavouring to ascertain the practicability of such treatment on the human subject and ultimately I think [it] will be found used with perfect safety and success in Surgical operations.<sup>13</sup> To this letter is appended an account of seven operations on animals rendered insensible either through the inhalation of carbonic acid gas or by the rebreathing of atmospheric air in a closed chamber.

Although Hickman experimented with different gases and was even credited many years later by a member of the Académie de Médecine in Paris<sup>14</sup> with using  $N_2O$  he refers only to carbonic acid gas. In the light of recent research on the role of  $CO_2$  in anæsthesia this choice was unfortunate. At the time Hickman failed to gain encouragement from the medical profession either in this country or in France. Ardently as he believed that inhalation anæsthesia would be as successful with men as with animals he hesitated unsupported by a single colleague to apply his experimental findings to man. Nevertheless he deserves recognition as the first to advocate unequivocally the principle of producing surgical anæsthesia by inhalation.

During the first half of the nineteenth century popular lectures of an instructive though non technical character on chemistry had an immense vogue in America. On 10th December 1844 at Hartford Connecticut the dentist Horace Wells (1815-1848) attended a lecture advertised as "A Grand Exhibition of the Effects produced by inhaling Nitrous Oxid. Exhilarating or Laughing Gas!" given by Gardner Quincy Colton (1814-1898) who had studied medicine but had never taken a medical degree. At the demonstration which followed and enlivened this dissertation<sup>15</sup> Wells's curiosity was aroused by the observation that a young man named Cooley who after inhaling  $N_2O$

had stumbled and barked his shins severely nevertheless seemed quite oblivious to the accident. Wells drew him aside and questioned him closely and finding that he stoutly maintained that he had not felt the slightest pain became much excited and said to a friend ' I believe a man by taking that gas could have a tooth extracted or a limb amputated and not feel any pain. Before leaving the hall he discussed the possibility with Colton and a dentist named Riggs and it was arranged that next day in the presence of witnesses, Wells should inhale nitrous oxide administered to him by Colton while Riggs pulled out one of his teeth. Anaesthesia in this instance proved an unqualified success.

During the next few weeks Wells assisted by Riggs extracted teeth from a number of people and in January 1845 through another dentist his former partner William Thomas Green Morton (1819-1868) obtained permission to demonstrate his discovery at the Massachusetts General Hospital in Boston. It was arranged by Dr J C Warren one of the leading surgeons of the hospital that Wells should first address a class of students and then demonstrate the extraction of a tooth from a young man under nitrous oxide. Full details of what happened at that demonstration will never be known. Wells seems to have acted as extractor an assistant administering the gas probably from a comparatively small bladder shaped rubber bag with a simple mouth tap. It is generally stated that the assistant withdrew the bag too soon. As the tooth came out the patient yelled lustily, though afterwards admitting that he felt little, if any pain. Poor Wells was accused of humbug and hooted out of the theatre by the students and Warren bothered no further with him<sup>15</sup>. Nitrous oxide was discredited and for eighteen years fell into disuse.

In explanation of the failure of Wells's demonstration, it may be surmised that the patient was of the robust type now described as anaesthetic resistant for under nitrous oxide such a patient becomes cyanosed before anaesthesia has been attained, and in those early days when cyanosis was doubtless taken as a danger-signal it is probable that administration would have been stopped in appreciable time before surgical anaesthesia had been attained. To this day it is sometimes impossible to produce perfectly tranquil narcosis, even for dental extractions if nitrous oxide is used to anaesthetise a robust male, particularly if he is nervous and unpremedicated.

Undeterred by Wells's failure his former partner Morton continued to investigate the possibility of painless extraction. Since like Wells he specialised in prosthetic dentistry the incentive to discover a means of relieving pain during extractions was strong. The idea of

admitted a member of the Royal College of Surgeons, and set up in practice in Shropshire. Although little older than Davy had been when he carried out the researches on nitrous oxide. Hickman differed from Davy in that he had the clearly defined object of showing that surgical anæsthesia could be achieved by inhalation. He formulated this belief in a pamphlet published in 1824<sup>12</sup>. In a letter addressed to his friend T. A. Knight F.R.S. he says: "There is not an individual who does not shudder at the idea of an operation" and I have frequently lamented when performing my own duties as a Surgeon that something has not been thought of whereby the fears may be tranquilised and suffering relieved. Above all from the many experiments on suspended Animation I have wondered that some hint has not been thrown out of its probable utility and noticed by Surgeons and consequently I have been induced to make experiments on Animals endeavouring to ascertain the practicability of such treatment on the human subject and ultimately I think [it] will be found used with perfect safety and success in Surgical operations<sup>13</sup>. To this letter is appended an account of seven operations on animals rendered insensible either through the inhalation of carbonic acid gas or by the rebreathing of atmospheric air in a closed chamber.

Although Hickman experimented with different gases and was even credited many years later by a member of the Académie de Médecine in Paris<sup>14</sup> with using  $N_2O$  he refers only to carbonic acid gas. In the light of recent research on the role of  $CO_2$  in anæsthesia this choice was unfortunate. At the time Hickman failed to gain encouragement from the medical profession either in this country or in France. Ardently as he believed that inhalation anæsthesia would be as successful with men as with animals he hesitated, unsupported by a single colleague, to apply his experimental findings to man. Nevertheless he deserves recognition as the first to advocate unequivocally the principle of producing surgical anæsthesia by inhalation.

During the first half of the nineteenth century popular lectures of an instructive though non technical character on chemistry had an immense vogue in America. On 10th December 1844 at Hartford Connecticut the dentist Horace Wells (1815-1848) attended a lecture advertised as "A Grand Exhibition of the Effects produced by inhaling Nitrous Oxid. Exhilarating or Laughing Gas"<sup>1</sup> given by Gardner Quincy Colton (1814-1898) who had studied medicine but had never taken a medical degree. At the demonstration which followed and enlivened this dissertation<sup>15</sup> Wells's curiosity was aroused by the observation that a young man named Cooley who after inhaling  $N_2O$

had stumbled and barked his shins severely nevertheless seemed quite oblivious to the accident. Wells drew him aside and questioned him closely and finding that he stoutly maintained that he had not felt the slightest pain became much excited, and said to a friend, 'I believe a man by taking that gas could have a tooth extracted or a limb amputated and not feel any pain'. Before leaving the hall he discussed the possibility with Colton and a dentist named Riggs and it was arranged that next day in the presence of witnesses Wells should inhale nitrous oxide administered to him by Colton while Riggs pulled out one of his teeth. Anaesthesia in this instance proved an unqualified success.

During the next few weeks Wells assisted by Riggs extracted teeth from a number of people and in January 1845 through another dentist his former partner William Thomas Green Morton (1819-1868) obtained permission to demonstrate his discovery at the Massachusetts General Hospital in Boston. It was arranged by Dr J. C. Warren one of the leading surgeons of the hospital, that Wells should first address a class of students and then demonstrate the extraction of a tooth from a young man under nitrous oxide. Full details of what happened at that demonstration will never be known. Wells seems to have acted as extractor an assistant administering the gas probably from a comparatively small bladder shaped rubber bag with a simple mouth tap. It is generally stated that the assistant withdrew the bag too soon. As the tooth came out the patient yelled lustily though afterwards admitting that he felt little if any, pain. Poor Wells was accused of humbug and hooted out of the theatre by the students, and Warren bothered no further with him.<sup>15</sup> Nitrous oxide was discredited and for eighteen years fell into disuse.

In explanation of the failure of Wells's demonstration, it may be surmised that the patient was of the robust type now described as anaesthetic resistant for under nitrous oxide such a patient becomes cyanosed before anaesthesia has been attained, and in those early days when cyanosis was doubtless taken as a danger-signal it is probable that administration would have been stopped in appreciable-time before surgical anaesthesia had been attained. To this day it is sometimes impossible to produce perfectly tranquil narcosis even for dental extractions if nitrous oxide is used to anaesthetise a robust male particularly if he is nervous and unpremedicated.

Undeterred by Wells's failure his former partner Morton continued to investigate the possibility of painless extraction. Since like Wells, he specialised in prosthetic dentistry the incentive to discover a means of relieving pain during extractions was strong. The idea of

trying ether probably suggested itself as a result of the frolics so popular in Europe and America during the first half of the nineteenth century at which small quantities of nitrous oxide or ether were inhaled for the feeling of exhilaration which they produced. At these parties it had been conclusively established not only that ether vapour was respirable but that too large a quantity caused stupor. Michael Faraday had pointed out in 1818 that in these respects the effects of ether and nitrous oxide were similar.<sup>16</sup>

As nitrous oxide had been discredited the distinction of discovering anæsthesia would belong to the one who introduced ether. In the bitter quarrel which immediately followed the successful use of ether Morton claimed that he had introduced it but Dr Charles Thomas Jackson (1805-1880) chemist and geologist alleged that the credit should be his since he had suggested its use to Morton. Weighing up the pros and cons of the argument it now seems reasonably certain that Jackson did not originally suggest the use of ether but it seems equally certain that he gave Morton valuable advice. For Morton having tried the inhalation of ether upon one or two household pets without mishap nevertheless admitted to his partner Grenville G. Hayden<sup>17</sup> who appears to be a fairly reliable witness that 'in some particulars his discovery did not work exactly right whereupon Hayden advised him to consult a chemist. Towards the end of September 1846 Morton saw Jackson and though he did not disclose that he had been experimenting with ether managed to glean important information. Dr Morton told me wrote Hayden in his testimony that he had just tried ether again—in accordance with Jackson's hint—on himself and that he had remained insensible seven or eight minutes by the watch. A few days later on 30th September 1846, Morton pouring ether on a folded cloth successfully anæsthetised Eben Frost for the extraction of a tooth. However all was not yet plain sailing. A series of failures followed and Morton once more consulted Jackson who this time recommended the use of a glass inhaler. After making certain modifications in the inhaler Morton demonstrated ether anæsthesia at the Massachusetts General Hospital on 16th October 1846. At this demonstration he administered ether under the name of *letheon* (trying unsuccessfully to keep its precise nature secret) and Warren dissected out a congenital but superficial vascular tumor from the neck of a man. As the patient recovered consciousness Warren exclaimed 'Gentlemen this is no humbug'.

It subsequently transpired that Crawford W. Long (1815-1878) a busy general practitioner had used ether as early as 1842 in a few minor surgical operations in Georgia U.S.A. but the exigencies of a

large practice and local apathy and opposition gave him neither opportunity nor encouragement to pursue his discovery further."

It is of some interest to note that it was Colton who reintroduced nitrous oxide as an anæsthetic. He had continued to travel about America giving popular lectures and after 1844 had included an account of Wells's discovery and of how the anæsthetic property of this gas had been made use of for tooth extraction. In 1862 after a lecture-demonstration at New Britain Connecticut, an old lady asked to be allowed to inhale the gas to have some teeth out for she was afraid both of ether and of chloroform. In contrast to the patient chosen for Wells's demonstration, this elderly co-operative woman is now recognised as being a suitable subject for displaying impressively the advantages of nitrous oxide. It is not surprising therefore that Colton with his previous wide experience of this gas, administered it to the satisfaction both of the patient and her dentist. Colton afterwards taught the dentist Dr Dunham, how to make the gas.

On his next lecture visit to the town a year later Colton found Dunham in a flourishing practice busily extracting teeth under nitrous-oxide anæsthesia. He immediately decided that in future the painless extraction of teeth should be a feature of his lecture-demonstrations.

In a few weeks wrote J Marion Sims in 1877 "people came by hundreds to take the gas and get teeth extracted. This experiment convinced Colton that it could be made a great business and he went to New York and opened the Colton Dental Institute, where, since 1863 he and his agents have given the gas to 97 000 persons without accident."

It is noteworthy that nitrous oxide and ether, the first two anæsthetics discovered have retained their popularity and that their use has not been seriously challenged despite the introduction of many other anæsthetics. By reason of its safety ether still remains the anæsthetic of choice for operations requiring muscular relaxation. For minor operations nitrous oxide has the advantage of being eliminated much more rapidly and of providing quick recovery.

Simpson (1811-1870) was the first to use chloroform as an anæsthetic. In a paper<sup>29</sup> to the Medico Chirurgical Society of Edinburgh on 10th November 1847 he wrote 'I am enabled to speak most confidently of its superior anæsthetic properties having now tried it upon upwards of thirty individuals.' His success with chloroform coupled with his enthusiastic championship of it quickly convinced the medical profession not only in Great Britain but also on the Continent, and to a certain extent even in America that chloroform was indeed far superior in every way to ether. This widespread belief produced

both evil and good evil because chloroform is intrinsically dangerous good because the recognition of those inherent dangers was more powerful than any other factor in promoting scientific investigation into the nature of anæsthesia and of anæsthetic agents

The first to appreciate the urgent need for physiological research and the man whose influence guided the subsequent course of anæsthesia for many years was the Yorkshireman John Snow (1813-1858) whose books On Ether and On Anæsthetics should be read by every anæsthetist It was Snow who set the seal of propriety on anæsthesia in obstetrics when in 1853 he administered chloroform to Queen Victoria during the birth of Prince Leopold and again in 1857 at the birth of Princess Beatrice Richardson<sup>21</sup> in 1858 writes Inquisitive folk often overburthened Snow after these events with a multitude of questions

One lady of an inquiring mind to whom he was administering chloroform got very loquacious during the period of excitement and declared she would inhale no more of the vapour unless she were told what the Queen said word for word when she was taking it Her Majesty replied the dry doctor asked no questions until she had breathed very much longer than you have and if you will only go on in loyal imitation I will tell you everything The patient could not but follow the example held out to her In a few seconds she forgot all about Queen Lords and Commons and when the time came for a renewal of hostilities found that her clever witness had gone home to his dinner leaving her with the thirst for knowledge still on her tongue It is noteworthy that paralysis of the intercostal muscles a sign of anæsthesia (p. 66) rediscovered by successive generations of anæsthetists was first described by Snow If the inhalation is continued the breathing is rendered difficult feeble or irregular and is sometimes performed only by the diaphragm whilst the intercostal muscles are paralysed<sup>22</sup> It was Snow too who first made use of an agent to absorb carbon dioxide in a closed circuit during anæsthesia Under these conditions he anæsthetised animals and further using chloroform and ether he rendered himself analgesic for long periods employing a solution of caustic potash as the absorbant<sup>23</sup>

At Snow's death his mantle descended upon the shoulders of Clover (1825-1882) He enjoyed a busy practice with the leading surgeons of his day, and spent most of his spare time in his workshop His notes sketches and experimental models presented in 1939 to the Department of Anæsthetics University of Oxford by his son and daughter show that he had in his mind ideas many years ahead of his time He wrote comparatively little and is best remembered by his ether-inhaler<sup>24</sup> and it should not be overlooked that it was he who

first suggested and made practicable a nitrous oxide ether sequence.<sup>25</sup> By this not only was the unpleasantness of an ether induction avoided but after anaesthesia had been established rapidly with  $N_2O$  the ether concentration in the inhaled mixture could be increased uniformly.

The technique for the administration of general anaesthetics advanced remarkably little until the twentieth century. The 1914-1918 War was responsible for the more general adoption of endotracheal anaesthesia. This method seems first to have been used by Trendelenburg in 1869. He realised the importance of plugging off the larynx to prevent the inspiration of blood and debris during extensive operations in the buccal cavity. Before the operation was commenced tracheotomy was performed and the administration of the anaesthetic continued through a cannula surrounded by an inflatable cuff inserted into the trachea through the wound.<sup>26</sup>

In 1878 William MacLewen, surgeon to the Glasgow Royal Infirmary, decided to essay the introduction of tracheal tubes by the mouth instead of performing tracheotomy or laryngotomy.<sup>27</sup> He carried out a number of experiments *post mortem* and found that instruments of the tube kind could after a little practice be passed with facility through the mouth into the trachea by introducing the finger into the mouth depressing the epiglottis on the tongue and so guiding the tube over the back of the finger into the larynx. His first case is described as Removal of epithelioma from pharynx and base of tongue introduction of tube into the trachea through mouth to occlude haemorrhage from larynx and for administration of anaesthetic. A large-bore flexible brass tube<sup>28</sup> was passed on the completely unanaesthetised patient and he writes "The usual cough ceased as he received a few whiffs of chloroform" the upper opening of the larynx was stuffed with a sponge.

Lyon after the introduction in 1895 of Kärstén's Autoscope<sup>29</sup> the first direct vision laryngoscope endotracheal tubes were passed by the sense of touch with the aid of a hook shaped pilot introducer or the larynx was exposed by a tongue spatula as advocated by the French surgeon Doyen (1859-1916). At this period sometimes general anaesthesia was induced more often the larynx was cocaineised before intubation.

Kuhn in 1902 further popularised laryngeal intubation. He advocated cocaineisation rather than general anaesthesia and he used a large bore flexible metal tube and a hook shaped introducer and resorted to exposing the larynx with a spatula only in difficult cases. It is interesting to note too that he administered anaesthetics under positive pressure gave chloroform in a  $CO_2$  absorption apparatus and



records three cases in which a tube was passed through the nose into the trachea <sup>30</sup>

In 1909 Meltzer and Auer physiologists, of the Rockefeller Institute New York became interested in the problem of continuous respiration without respiratory movement. They carried out a number of experiments on dogs insufflating a continuous current of oxygen laden with ether vapour through a small endotracheal catheter allowing the insufflated air to escape by way of the space left between the wall of the catheter and the vocal cords. In reporting these experiments Meltzer says: Another important and practical feature which we have observed in this method is its relation to anæsthesia.

It seems to me that the giving of ether by the method of intratracheal insufflation is the safest and most effective way of administration of this anæsthetic <sup>31</sup>

Meltzer's idea was adapted to clinical anæsthesia by Elsberg <sup>3</sup> also of New York who in passing the tubes made use of Chevalier Jackson's laryngoscope as a routine measure although many of his followers still preferred to use introducers. Although Elsberg's work attracted widespread attention endotracheal anæsthesia continued to be used on rare occasions only and the passing of a tube was regarded as a major procedure. It was not until anæsthetists were faced with the unprecedented problems of war mutilated soldiers needing extensive plastic surgery of the head and face that endotracheal anæsthesia was widely adopted. To Rowbotham and Magill <sup>32</sup> must be given the credit for so simplifying the technique that endotracheal anæsthesia has become generally adopted for a wide variety of operations.

The psychological advantages which accrue from producing unconsciousness before an inhalation anæsthetic is administered appear to have been appreciated first by the French naval surgeon Forné who in 1874 employed doses of 2-5 g. of chloral for this purpose <sup>34</sup>. Later intravenous ether <sup>35</sup> and intravenous hedonal <sup>36</sup> were used but it was not until 1913 when Gwathmey induced anæsthesia by giving a mixture of ether and olive oil rectally <sup>37</sup> that the method was considered simple enough and the results sufficiently constant and safe for the principle to become established. In 1926 Willstätter and Duisberg, searching for a remedy which could be given rectally to abolish the spasm of whooping-cough <sup>38</sup> introduced a new drug tribromethylalcohol now known as Avertin. It was at once recognised by Fritz Eichholtz <sup>39</sup> as an extremely promising anæsthetic. After being widely tested as such in Germany and at Eichholtz's request in England, <sup>40</sup> it rapidly attained wide popularity and still remains one of the most useful of basal anæsthetics.

Since 1921 various derivatives of barbituric acid have been used as basal anesthetics with varying success but this group of drugs did not attain universal popularity until 1932, when B. Weese<sup>11</sup> discovered the ultra short acting barbiturate, named Evipan. Weese confirmed by experiment the value of evipan not only as a rapidly acting hypnotic but as a total anesthetic and in 1933 Wilhelm Bretzner<sup>12</sup> of Berlin reported 400 surgical cases in which he had used evipan successfully either as a basal or as the sole anesthetic.

In the last two decades there have been great advances and refinements in anesthesia. In the 1940s the barbiturates found an established place and the introduction of cyclopropane and of the carbon dioxide absorption technique were two outstanding contributions of Ralph M. Waters of Madison U.S.A. In 1937, through the munificence of Lord Nuffield the first Chair of Anesthetics was founded in this country. The 1940s have been noteworthy for the increasing popularity of curarising drugs following Griffith's publication in 1942<sup>13</sup> and it is entertaining to reflect that the ancient and mysterious South American arrow poison figures in its modernised standardised stabilised form as the latest blessing of twentieth century anesthesia.

#### REFERENCES

- (1) Thompson R. Campbell 1924 *The Assyrian Herbal* London 43
- (2) Homer *The Odyssey* Bk. 4. I. Caulfield transl. 1923 London 51
- (3) Hort A. 1916 *Theophrastus* London 2, 259
- (4) Bostock J. and Riley H. T. 1854 *The Natural History of Pliny* London 5, 138
- (5) Gunther R. T. 1934 *The Greek Herbal of Dioscorides* Oxford 473-622
- (6) For examples see Paré A. 1634 *Works* London
- (7) Wedel G. W. 1682 *Opiologia* Jena 129
- (8) Serturmer I. W. A. 1806 *J. d. Pharmacie sur Aer.le* 14, 87
- (9) Magendie F. 1836 *A Formulary for the Preparation of Certain New Remedies* London
- (10) Beddoes T. 1796 *Considerations on the Medicinal Use of Factitious Airs* Bristol
- (11) Davy H. 1800 *Researches Chemical and Philosophical* London
- (12) Hickman H. H. 1824 *A Letter on Suspended Animation* Ironbridge
- (13) Hickman H. H. 1824 *Letter to T. A. Knight* Original in the Wellcome Museum London
- (14) Report of Meeting 1846/47 *Bull. Acad. Roy. Méd. Paris* 12, 396
- (15) Wells C. J. 1935 *Anesth. and Analges.* 14, 176-216
- (16) Article attributed to Faraday M. 1818 *J. Sci. and Arts* London 4, 158
- (17) Dana R. H. 1848 *Littell's Living Age* No. 201, 529
- (18) Taylor F. Long 1928 *Crawford W. Long* New York
- (19) Sims J. M. 1877 *Virginia med. Mon.* 4, 81

records three cases in which a tube was passed through the nose into the trachea <sup>30</sup>

In 1909 Meltzer and Auer physiologists, of the Rockefeller Institute New York became interested in the problem of continuous respiration without respiratory movement. They carried out a number of experiments on dogs insufflating a continuous current of oxygen laden with ether vapour through a small endotracheal catheter, allowing the insufflated air to escape by way of the space left between the wall of the catheter and the vocal cords. In reporting these experiments Meltzer says: "Another important and practical feature which we have observed in this method is its relation to anæsthesia."

It seems to me that the giving of ether by the method of intra tracheal insufflation is the safest and most effective way of administration of this anæsthetic <sup>31</sup>

Meltzer's idea was adapted to clinical anæsthesia by Elsberg <sup>3</sup> also of New York who in passing the tubes made use of Chevalier Jackson's laryngoscope as a routine measure although many of his followers still preferred to use introducers. Although Elsberg's work attracted widespread attention endotracheal anæsthesia continued to be used on rare occasions only and the passing of a tube was regarded as a major procedure. It was not until anæsthetists were faced with the unprecedented problems of war-mutilated soldiers needing extensive plastic surgery of the head and face that endotracheal anæsthesia was widely adopted. To Rowbotham and Magill <sup>33</sup> must be given the credit for so simplifying the technique that endotracheal anæsthesia has become generally adopted for a wide variety of operations.

The psychological advantages which accrue from producing unconsciousness before an inhalation anæsthetic is administered appear to have been appreciated first by the French naval surgeon Forné who in 1874 employed doses of 2-5 g. of chloral for this purpose <sup>34</sup>. Later intravenous ether <sup>35</sup> and intravenous hedonal <sup>36</sup> were used but it was not until 1913 when Gwathmey induced anæsthesia by giving a mixture of ether and olive oil rectally <sup>37</sup> that the method was considered simple enough and the results sufficiently constant and safe for the principle to become established. In 1926 Willstätter and Duisberg searching for a remedy which could be given rectally to abolish the spasm of whooping-cough <sup>38</sup> introduced a new drug tribromomethylalcohol now known as Avertin. It was at once recognised by Fritz Eichholtz <sup>39</sup> as an extremely promising anæsthetic. After being widely tested as such in Germany and at Eichholtz's request in England <sup>40</sup> it rapidly attained wide popularity and still remains one of the most useful of basal anæsthetics.

## CHAPTER II

### GENERAL REMARKS

Surgical anesthesia may be either local, regional, spinal or general.

1 **Local** — The drug in liquid form is applied to areas of mucous membrane e.g. of the nose or throat or it is injected into the immediate neighbourhood of the site of operation e.g. the mucous membrane and deep tissues around a tooth. Local anesthesia or insensitivity results from the narcotisation of afferent nerve endings.

2 **Regional** — A solution of the drug is injected into the immediate neighbourhood of a selected sensory nerve, and by direct action on the nerve fibres blocks transmission of impulses from the region which this nerve supplies e.g. the arm can be rendered insensitive to pain by an injection made above the clavicle which reaches the brachial plexus, and an injection into the neighbourhood of the inferior dental nerve as it is about to enter its foramen produces anesthesia of the mandible on that side.

Local anesthesia may be combined with regional. For an abdominal operation the skin and subcutaneous tissues may be anesthetised by local infiltration while flaccidity of the abdominal muscles and insensitivity of the peritoneum may be obtained by injecting the anæsthetic around the lower intercostal nerves where they enter the muscles.

3 **Spinal** — The anæsthetic solution is injected intrathecally (i.e. into the spinal canal) and there mixes with the cerebrospinal fluid bathing the nerves inside the canal. The spinal cord terminates at the lower border of the first lumbar vertebra and to avoid the danger of injuring it the injection is made below this level usually between the second and third lumbar vertebra. The object of spinal anesthesia is to block the sensory nerves of several segments of the cord although the corresponding motor nerves are necessarily also involved. Since the nerve roots in this situation are very close together, many can be affected simultaneously and a large area of the body thus anesthetised by a single injection. The distance to which the anæsthetic spreads in the spinal canal is determined by the amount of anæsthetic injected by the patient's posture and by the specific gravity of the injected solution. Intrathecal injection is rarely employed for operations.

- (20) Simpson J Y 1847 *Account of a New Anæsthetic Agent* Edinburgh  
 (21) Snow J 1858 *On Chloroform and Other Anæsthetics* London  
 XXXI  
 (22) Snow J *Ibid* 42  
 (23) Snow J 1850 *Lond med Gaz* 11 749  
*Ibid* 12, 622  
 (24) Clover J T 1877 *Brit med J* 1, 69  
 (25) Clover J T 1871 *Med Tms and Gaz* 2 603 1876 *Brit med J* 2 74  
 (26) Junker F 1872 *Med Tms and Gaz* 1, 510  
 (27) MacEwen W 1880 *Brit med J* 2, 122 164  
 (28) Primrose W 1940 Personal communication  
 (29) Kirstein A 1895 *Dtsch med Wschr* 38, 634  
 (30) Kuhn F 1905 *Dtsch Z Chir* 76, 148 1906 *Ibid* 81, 63  
 (31) Meltzer S J 1910 *Med Rec N Y* 77, 477  
 (32) Elsberg C A 1910 *Berl klin Wschr* 21 957  
 (33) Rowbotham E S and Magill I 1921 *Proc R Soc Med* 14 (1)  
 Section of Anæsthetics 17  
 (34) Lannelongue 1874 *Bull Soc Chirurgie Paris* 3, 619  
 (35) Burkhardt L 1909 *Arch exp Path Pharmac* 61 323  
 (36) Leading article 1912 *Brit med J* 1 1378  
 (37) Gwathmey J T 1914 *Proc 17th International Congress of Medicine*  
 Subsection 7 (B) London 163  
 (38) Weese H 1937 Personal communication  
 (39) Eichholtz F 1927 *Dtsch med Wschr* 53 710  
 (40) Blomfield J and Shipway F E 1929 *Lancet* 1, 546  
 (41) Weese H 1932 *Dtsch med Wschr* 2 1205  
 (42) Baetzner W 1933 *Dtsch med Wschr* 59, 48  
 (43) Griffith H R and Johnson G E 1942 *Anesthesiology* 3, 418

the following considerations. That—

Anæsthesia is a pathological state and its production is a serious matter not to be undertaken lightly

Any drug which produces the state of unconsciousness with freedom known as anæsthesia is a poison although in the case of the anæsthetics in common use the poison is controllable and its action reversible

Patients cannot be rendered unconscious by any means without doing them some general harm and involving them in a certain if only a minute degree of risk

Anæsthetics should be given only when it is believed that the resulting relief will more than compensate for their poisonous effects

The anæsthetics to be employed must cause the minimum of damage

Only those drugs which have stood the test of time should be in routine use for the production of anæsthesia

To merit inclusion in the list of recognised anæsthetic agents a drug must have the following advantages over other means of producing unconsciousness

1 *Its Action must be Reversible*—The anæsthetic administered must be either excreted or metabolised rapidly enough to allow the patient to recover within a reasonable time

2 *Its Action must be Controllable*—With *inhalation anæsthetics* depth of anæsthesia is controlled by regulating the strength of the mixture delivered to the patient. Certain changes in respiratory rhythm foretell the approach of dangerously deep anæsthesia and give the watchful administrator ample time to discontinue the anæsthetic by removing the face piece. Even if these warnings are ignored and the supply of anæsthetic is maintained the diminution of the respiratory excursions reduces the intake of the drug and respiratory arrest ensues immediately the amount of anæsthetic in the blood stream reaches the level which causes this condition. A rise above this level is impossible since intake of the drug ceases with respiratory arrest. If removal of the mask has been delayed until this emergency has supervened artificial respiration if instituted promptly will eliminate sufficient anæsthetic via the lungs to reduce the concentration in the blood stream below that which has paralysed the respiratory centre. That elimination has been effective is shown by the almost immediate resumption of normal respiration. Maintenance of any desired level of anæsthesia is effected most safely and easily with inhalation anæsthetics since these are more controllable than those given by other routes. This is of much importance where profound anæsthesia is

necessitating anæsthesia above the level of the area supplied by nerves emerging from the fourth dorsal segment of the spinal cord. If anæsthesia of higher regions is attempted by this means the injected fluid is liable to spread to the level of the fifth cervical segment and paralyse the phrenic nerve which supplies the diaphragm. This would bring respiration to a standstill because by the time the anæsthetic reaches this level the diaphragm would be the only respiratory muscle still functioning the intercostal nerves having been already affected during the upward spread of the anæsthetic.

**4 General**—The patient is rendered unconscious and for practical purposes unresponsive to stimuli applied to any part of the body—hence the term 'general'. This is the only type of anæsthesia considered in this book. The drug is distributed by the blood-stream through the whole body and anæsthesia results from its action on the brain. The anæsthetic may be administered either by (a) inhalation (b) intravenous injection (c) mouth (d) rectum less commonly by (e) subcutaneous injection or (f) intramuscular injection.

Experience has shown that only certain drugs are suitable for the production of general anæsthesia common examples being nitrous oxide ether and thiopentone the chemical compositions of which differ widely.

Natural sleep is a physiological state of unconsciousness and differs from anæsthesia in that a patient reacts to stimuli and is easily roused to consciousness. By the use of less than a full dose of any anæsthetic a patient can be rendered unconscious but, according to the amount given, may react excessively or sluggishly to a painful stimulus. The response to such a stimulus is excessive if sufficient anæsthetic is given to remove only the patient's higher conscious control and becomes progressively less as larger doses are administered. Such unconsciousness accompanied by amnesia for pain but not by loss of reflex action, is unsuitable for operations because the patient's reactions may interfere with the surgeon's work.

A state indistinguishable from general anæsthesia occurs also as a result of poisoning e.g. by coal gas or bacterial toxins (such as from typhoid fever or diphtheria) the toxins of uræmia or diabetes large doses of alcohol morphia aspirin etc. Extremes of heat and cold or severe trauma or partial drowning may also result in a similar condition. Unconsciousness from any of these factors or indeed the moribund state preceding death is often indistinguishable from one or other of the stages of anæsthesia produced by recognised general anæsthetics. Realisation of this fact emphasises the importance of

the following considerations That—

Anæsthesia is a pathological state and its production is a serious matter not to be undertaken lightly

Any drug which produces the state of unconsciousness with flaccidity, known as anæsthesia is a poison, although in the case of the anæsthetics in common use the poison is controllable and its action reversible

Patients cannot be rendered unconscious by any means without doing them some general harm and involving them in a certain, if only a minute, degree of risk

Anæsthetics should be given only when it is believed that the resulting relief will more than compensate for their poisonous effects

The anæsthetics to be employed must cause the minimum of damage

Only those drugs which have stood the test of time should be in routine use for the production of anæsthesia

To merit inclusion in the list of recognised anæsthetic agents, a drug must have the following advantages over other means of producing unconsciousness

1 *Its Action must be Reversible* —The anæsthetic administered must be either excreted or metabolised rapidly enough to allow the patient to recover within a reasonable time

2 *Its Action must be Controllable* —With *inhalation anæsthetics* depth of anæsthesia is controlled by regulating the strength of the mixture delivered to the patient. Certain changes in respiratory rhythm foretell the approach of dangerously deep anæsthesia and give the watchful administrator ample time to discontinue the anæsthetic by removing the face-piece. Even if these warnings are ignored and the supply of anæsthetic is maintained, the diminution of the respiratory excursions reduces the intake of the drug and respiratory arrest ensues immediately the amount of anæsthetic in the blood stream reaches the level which causes this condition. A rise above this level is impossible since intake of the drug ceases with respiratory arrest. If removal of the mask has been delayed until this emergency has supervened artificial respiration if instituted promptly will eliminate sufficient anæsthetic via the lungs to reduce the concentration in the blood-stream below that which has paralysed the respiratory centre. That elimination has been effective is shown by the almost immediate resumption of normal respiration. Maintenance of any desired level of anæsthesia is effected most safely and easily with inhalation anæsthetics since these are more controllable than those given by other routes. This is of much importance where profound anæsthesia is



necessitating anæsthesia above the level of the area supplied by nerves emerging from the fourth dorsal segment of the spinal cord. If anæsthesia of higher regions is attempted by this means the injected fluid is liable to spread to the level of the fifth cervical segment and paralyse the phrenic nerve which supplies the diaphragm. This would bring respiration to a standstill because by the time the anæsthetic reaches this level the diaphragm would be the only respiratory muscle still functioning the intercostal nerves having been already affected during the upward spread of the anæsthetic.

**4 General**—The patient is rendered unconscious and for practical purposes unresponsive to stimuli applied to any part of the body—hence the term general. This is the only type of anæsthesia considered in this book. The drug is distributed by the blood stream through the whole body and anæsthesia results from its action on the brain. The anæsthetic may be administered either by (a) inhalation (b) intravenous injection (c) mouth (d) rectum less commonly by (e) subcutaneous injection, or (f) intramuscular injection.

Experience has shown that only certain drugs are suitable for the production of general anæsthesia common examples being nitrous oxide ether and thiopentone the chemical compositions of which differ widely.

Natural sleep is a physiological state of unconsciousness and differs from anæsthesia in that a patient reacts to stimuli and is easily roused to consciousness. By the use of less than a full dose of any anæsthetic a patient can be rendered unconscious but, according to the amount given, may react excessively or sluggishly to a painful stimulus. The response to such a stimulus is excessive if sufficient anæsthetic is given to remove only the patient's higher conscious control and becomes progressively less as larger doses are administered. Such unconsciousness accompanied by amnesia for pain but not by loss of reflex action, is unsuitable for operations because the patient's reactions may interfere with the surgeon's work.

A state indistinguishable from general anæsthesia occurs also as a result of poisoning, e.g. by coal gas or bacterial toxins (such as from typhoid fever or diphtheria) the toxins of uræmia or diabetes large doses of alcohol morphia aspirin etc. Extremes of heat and cold or severe trauma or partial drowning may also result in a similar condition. Unconsciousness from any of these factors or indeed the moribund state preceding death is often indistinguishable from one or other of the stages of anæsthesia produced by recognised general anæsthetics. Realisation of this fact emphasises the importance of

of an accident or error of judgment. For example in the use of paraldehyde which is frequently given rectally as a basal anæsthetic in doses of 5j per stone of body weight confusion between the symbols for the drachm (ʒ) and the ounce (5) is known to have led to the administration of eight times the intended dose with fatal results.

If it should be found that an overdose has been given any drug remaining unabsorbed in the stomach may be recovered by gastric lavage. Similarly, an attempt should be made by washing out the rectum to recover substances administered by that route, but in practice it is found to be more difficult to recover a drug from the rectum than from the stomach.

3 *Its Action must be Predictable*—Within reasonable limits an anæsthetist can foretell the effects of a given quantity of thiopentone on a given patient. Rate of recovery varies with different anæsthetics but depends in great part on the dosage. Experience and a knowledge of the amount administered enable one to predict approximately the time of recovery.

4 *The Therapeutic Quotient must be High* i.e. the ratio between the minimum lethal dose and the minimum dose which must be given to produce unconsciousness must be high. The therapeutic quotient of hexobarbitone for instance is 4. This means that if 0.3 g is necessary to render a given patient unconscious it is unlikely that hexobarbitone will be lethal to that patient unless a dose of four times that quantity (1.2 g) is administered at one time.

5 *Its Side-actions should be Minimal*—Beyond the production of anaesthesia the physiological functions of the patient should be disturbed as little as possible. To achieve this the anæsthetist must select the drug most appropriate to the patient and for the operation and must have had experience in its use. Inhalation of nitrous oxide for a few minutes will not produce appreciable after-effects but the longer the administration the more probable will be the occurrence of nausea vomiting and even collapse. Ether a more powerful poison than nitrous oxide produces deeper anaesthesia and when its administration is discontinued is followed by a longer period of unconsciousness and by greater toxic effects on the body. These are frequently manifested by nausea and vomiting and occasionally by more serious disturbances such as post-anæsthetic albuminuria.

As already mentioned unconsciousness deep enough to permit the performance of a surgical operation may be produced by a wide variety of causes. It is possible to conceive of a classification of such causes arranged in order of merit on the basis of their degree of

required since at this level any excess of anæsthetic soon produces respiratory depression

The effects of drugs given intravenously (e.g. thiopentone) cannot be controlled so readily. It is true that anæsthesia can be deepened to any degree by the simple process of injection but the ease and rapidity with which an anæsthetic drug can be introduced into the blood stream is associated with the danger that an overdose may be given before the usual warning respiratory signs are manifest. With rapid injection although the onset of respiratory depression and arrest is sudden it is dangerously easy to introduce sufficient anæsthetic to raise the blood concentration considerably beyond that which causes respiratory arrest. Most intravenous anæsthetics are detoxicated by the liver and excreted through the kidneys and elimination is slow. Moreover if the blood concentration has been raised above that which produces respiratory arrest artificial respiration will have to be continued until the concentration is reduced to the level compatible with normal respiration. If the excess is slight artificial respiration will probably be needed for only a few minutes whereas if the excess is great enough artificial respiration will be unavailing no matter how long it is continued. In between these two extremes will be found cases where lives are saved by prolonged artificial respiration.

The precision with which it is possible to control the action of drugs given by mouth or rectum is even less than is the case with drugs which are given intravenously. Continuous administration by these routes is impracticable so that the whole of the estimated dosage is introduced while the patient is still conscious. Absorption from the rectum is more uniform than that of drugs given by mouth which depends on the general condition of activity of the alimentary tract which in turn may be considerably delayed by emotional states such as fear and excitement. More important however than the uncertainty as to the rate of absorption is the overriding consideration (applicable also to drugs injected intravenously) that if an overdose should be administered by either route absorption once it has begun will continue even after respiratory failure has occurred. For this reason it is not practicable to induce full surgical anæsthesia with substances given by mouth or rectum. They may be used only to bring about the state of basal anæsthesia which requires to be deepened further by the administration of a more controllable drug (e.g. a volatile anæsthetic) in order to carry out a surgical operation. The lack of controllability of drugs given by these routes enforces a deliberate avoidance of the use of doses large enough to be dangerously unmanageable which means that overdosage occurs only as the result

of an accident or error of judgment. For example in the use of paraldehyde which is frequently given rectally as a basal anæsthetic in doses of 5j per stone of body weight, confusion between the symbols for the drachm (ʒ) and the ounce (℥) is known to have led to the administration of eight times the intended dose with fatal results.

If it should be found that an overdose has been given any drug remaining unabsorbed in the stomach may be recovered by gastric lavage. Similarly, an attempt should be made by washing out the rectum to recover substances administered by that route, but in practice it is found to be more difficult to recover a drug from the rectum than from the stomach.

3 *Its Action must be Predictable*—Within reasonable limits an anæsthetist can foretell the effects of a given quantity of thiopentone on a given patient. Rate of recovery varies with different anæsthetics but depends in great part on the dosage. Experience and a knowledge of the amount administered enable one to predict approximately the time of recovery.

4 *The Therapeutic Quotient must be High* i.e. the ratio between the minimum lethal dose and the minimum dose which must be given to produce unconsciousness must be high. The therapeutic quotient of hexobarbitone for instance is 4. This means that if 0.3 g is necessary to render a given patient unconscious it is unlikely that hexobarbitone will be lethal to that patient unless a dose of four times that quantity (1.2 g) is administered at one time.

5 *Its Side-actions should be Minimal*—Beyond the production of anaesthesia the physiological functions of the patient should be disturbed as little as possible. To achieve this the anæsthetist must select the drug most appropriate to the patient and for the operation and must have had experience in its use. Inhalation of nitrous oxide for a few minutes will not produce appreciable after-effects but the longer the administration the more probable will be the occurrence of nausea vomiting and even collapse. Ether a more powerful poison than nitrous oxide produces deeper anaesthesia and when its administration is discontinued is followed by a longer period of unconsciousness and by greater toxic effects on the body. These are frequently manifested by nausea and vomiting and occasionally by more serious disturbances such as post anaesthetic albuminuria.

As already mentioned unconsciousness deep enough to permit the performance of a surgical operation may be produced by a wide variety of causes. It is possible to conceive of a classification of such causes arranged in order of merit on the basis of their degree of

reversibility controllability, and predictability, therapeutic quotient and intensity of side reactions. These causes would then form a continuous series of gradually decreasing safety, utility, and convenience. In practice doubtless there would be considerable disagreement over the exact order, few would care to assess the relative merits of alcohol and paraldehyde as means of producing surgical anæsthesia. Nevertheless the right of the anæsthetics in routine use to stand at the head of the list can hardly be disputed, and the last place would perhaps be generally assigned to that pre mortal coma which heralds the end in various toxic and traumatic conditions. This hypothetical list includes a group of pre-anæsthetic drugs so called because they are administered before induction of anæsthesia to assist the action of anæsthetics. They may be regarded as intermediate in their action between the recognised anæsthetics and the grosser causes of production of unconsciousness. These connecting links include morphia alcohol and paraldehyde which if exploited to their full extent can serve without assistance as adequate anæsthetics. These drugs are dealt with in the chapter entitled Pre anæsthetic Medication (p 88) but are mentioned here in order, on the one hand to emphasise the existence of a large number of compounds capable of producing anæsthesia which because of their gradually decreasing safety are of only academic interest to the practical anæsthetist and on the other to reiterate that there do not exist providentially provided substances whose sole action on the body is the production of anæsthesia.

## CHAPTER III

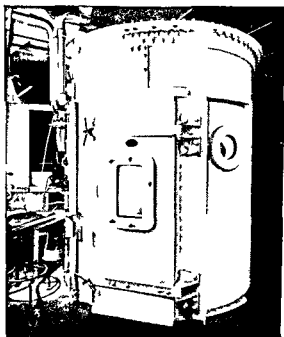
### THEORY OF ANÆSTHESIA

How do anæsthetics work? This is one of the first questions the student asks when he is introduced to the practice and problems of anæsthesia. That the correct answer is still a subject for speculation is shown by the allocation of more than thirty pages of Gwathmey's text book to the discussion of nineteen theories. Many additional theories are to be found elsewhere some intelligible only to their propounders.

Various drugs modify the actions of the different cells throughout the body. Those used as anæsthetics are chosen because they depress reversibly the activity of the central nervous system without causing generalised physiological disturbance of practical importance. A case can be made for the hypothesis that all phenomena desirable and otherwise of anæsthesia are attributable to anoxia. All the cells of the body require adequate oxygen for the continuance of their normal functions. Mental impairment and unconsciousness result from progressive oxygen lack. Production of unconsciousness by diminution of oxygen tension is exemplified when the airman loses consciousness on reaching great altitudes and regains it when his aeroplane falls to a level where the oxygen tension is sufficient to support mental activity. Different individuals lose consciousness at different levels of oxygen tension some at a level little reduced from the normal. Loss of consciousness at low oxygen tension can be demonstrated to a class by a volunteer inhaling an inert gas such as nitrogen or helium. In the average individual when the alveolar oxygen tension is reduced below approximately 30 mm Hg unconsciousness supervenes. Another method is to place the subject in a large steel decompression chamber (fig. 1) and to lower the atmospheric pressure within. When the pressure of air inside the cylinder is reduced to about 300 mm Hg (equivalent to an alveolar oxygen tension of 28 mm Hg) the patient will lose consciousness. A similar result may be achieved by administering a mixture of nitrogen 92 per cent and oxygen 8 per cent at normal atmospheric pressure.

The warning on p. 339 against experimenting with anæsthetics unless another person is present applies with equal force to experiments on the effects of low O<sub>2</sub> tension. In both cases the onset of

unconsciousness is insidious and fatalities have been recorded where this precaution has not been taken



RAF Officer Coulpygher d

FIG 1—Decompression Chamber

The way in which body cells utilise oxygen is extremely complex and is not yet fully understood. It is known however that during the oxidative processes many different compounds are formed. At each link in the complex chain of compounds oxygen transfer is mediated by an enzyme. If any one of these many enzymes is poisoned the oxygen transfer cannot take place with the result that the cell is deprived of the oxygen it needs in order to function normally. It has been shown that unconsciousness will result if the tension of oxygen to which the tissues are exposed is

reduced below a certain level. It is possible therefore that the action of anæsthetics is to produce a diminished intracellular oxygen tension despite a normal blood oxygen tension. How this is brought about is not known but it is not unreasonable to postulate that the various anæsthetics act by combining with the cell lipoids thus poisoning one or more of the many enzymes taking place in the complicated process of cell oxidation. The result will be that all cells which have absorbed an anæsthetic will lose their capacity to utilise the oxygen circulating in the bloodstream in proportion to the amount and potency of the drug they have absorbed. This hypothesis is supported by the fact that the amount of oxygen which the tissues consume becomes less and less as anæsthesia deepens although whether this is *post hoc* or *propter hoc* is not proven.

It must not be thought that the oxidative processes are entirely abolished by anæsthetics. If this were to happen for any length of time cell death would take place. This is seen in death from asphyxia following carbon monoxide poisoning where the oxygen-carrying

capacity of the blood is reduced to less than vital limits. It also occurs when an over dose of an anæsthetic completely abolishes intracellular oxidation. In surgical anæsthesia the oxidative processes are only diminished, and since the action of anæsthetics is reversible complete recovery of cell function takes place after the drug is eliminated.

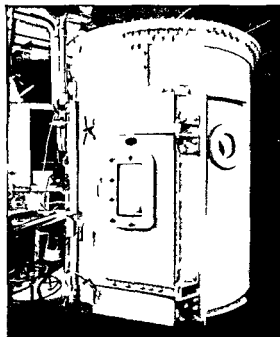
The oxygen deprivation theory of anæsthesia is bound up with the fact that anæsthetics are readily absorbed by fats. If an anæsthetic is added to a mixture of equal parts of olive oil and water it will be found, after the whole has been well shaken and allowed to stand that when the olive oil and water separate they will have taken up the anæsthetic in definite proportions. Similarly the amount of anæsthetic taken up by cell lipoids bears a constant ratio to that remaining in the blood-stream. In general the greater the affinity of the anæsthetic for fats the greater is its potency.

The functions of different cells of the body as a whole and indeed of any particular organ diminish at progressive stages of oxygen lack. The various nerve cells themselves differ in their reaction to  $O_2$ -lack. The more recent the phylogenetic development of a structure the more sensitive it is to  $O_2$ -lack, and the more liable it is to irreparable changes if this lack is complete or prolonged. Of the parts of the brain the more recently and highly developed cerebrum reacts to  $O_2$ -lack at an early stage so that rational thinking and consciousness are lost early. This is quickly followed by cerebellar disturbance. These two changes are familiar occurrences in drunkenness which can be regarded as being the first stage of anæsthesia by alcohol. In deep anæsthesia the processes of metabolism and elimination i.e. the functions of the liver and kidneys may be almost entirely suspended while primitive functions of respiration and cardiac action continue relatively unimpaired. Further deepening of anæsthesia is followed in turn by respiratory depression cessation of respiration and finally cardiac arrest.

There are two ways of reducing the oxygen available to the tissues in order to produce unconsciousness and anæsthesia. One is by the simple expedient of inhaling an inert gas such as nitrogen with the addition of just sufficient oxygen to support essential metabolic activity the other is to supply the patient with the usual (or even excess) amount of oxygen but at the same time to poison the cells so that they cannot utilise it in the normal way. As Macklin<sup>1</sup> puts it, there are two ways of starving a patient (i) simply by depriving him of food (the analogy is diminution of the oxygen content of the blood as in nitrous-oxide anæsthesia) and (ii) by giving him abundant



unconsciousness is insidious and fatalities have been recorded where this precaution has not been taken



RAF Officers Copyright  
FIG 1—Decompression Chamber

The way in which body cells utilise oxygen is extremely complex and is not yet fully understood. It is known however that during the oxidative processes many different compounds are formed. At each link in the complex chain of compounds oxygen transfer is mediated by an enzyme. If any one of these many enzymes is poisoned the oxygen transfer cannot take place with the result that the cell is deprived of the oxygen it needs in order to function normally. It has been shown that unconsciousness will result if the tension of oxygen to which the tissues are exposed is

reduced below a certain level. It is possible therefore that the action of anæsthetics is to produce a diminished intracellular oxygen tension despite a normal blood oxygen tension. How this is brought about is not known but it is not unreasonable to postulate that the various anæsthetics act by combining with the cell lipoids thus poisoning one or more of the many enzymes taking place in the complicated process of cell oxidation. The result will be that all cells which have absorbed an anæsthetic will lose their capacity to utilise the oxygen circulating in the bloodstream in proportion to the amount and potency of the drug they have absorbed. This hypothesis is supported by the fact that the amount of oxygen which the tissues consume becomes less and less as anæsthesia deepens although whether this is *post hoc* or *propter hoc* is not proven.

It must not be thought that the oxidative processes are entirely abolished by anæsthetics. If this were to happen for any length of time cell death would take place. This is seen in death from asphyxia following carbon monoxide poisoning where the oxygen carrying

capacity of the blood is reduced to less than vital limits. It also occurs when an over-dose of an anæsthetic completely abolishes intracellular oxidation. In surgical anæsthesia the oxidative processes are only diminished and since the action of anæsthetics is reversible complete recovery of cell function takes place after the drug is eliminated.

The oxygen deprivation theory of anæsthesia is bound up with the fact that anæsthetics are readily absorbed by fats. If an anæsthetic is added to a mixture of equal parts of olive oil and water it will be found after the whole has been well shaken and allowed to stand, that when the olive oil and water separate they will have taken up the anæsthetic in definite proportions. Similarly, the amount of anæsthetic taken up by cell lipoids bears a constant ratio to that remaining in the blood stream. In general the greater the affinity of the anæsthetic for fats the greater is its potency.

The functions of different cells of the body as a whole and indeed of any particular organ diminish at progressive stages of oxygen-lack. The various nerve cells themselves differ in their reaction to  $O_2$ -lack. The more recent the phylogenetic development of a structure the more sensitive it is to  $O_2$ -lack, and the more liable it is to irreparable changes if this lack is complete or prolonged. Of the parts of the brain the more recently and highly developed cerebrum reacts to  $O_2$ -lack at an early stage so that rational thinking and consciousness are lost early. This is quickly followed by cerebellar disturbance. These two changes are familiar occurrences in drunkenness, which can be regarded as being the first stage of anæsthesia by alcohol. In deep anæsthesia the processes of metabolism and elimination i.e. the functions of the liver and kidneys may be almost entirely suspended while primitive functions of respiration and cardiac action continue relatively unimpaired. Further deepening of anæsthesia is followed in turn by respiratory depression, cessation of respiration and finally cardiac arrest.

There are two ways of reducing the oxygen available to the tissues in order to produce unconsciousness and anæsthesia. One is by the simple expedient of inhaling an inert gas such as nitrogen with the addition of just sufficient oxygen to support essential metabolic activity the other is to supply the patient with the usual (or even excess) amount of oxygen but at the same time to poison the cells so that they cannot utilise it in the normal way. As Macklin<sup>1</sup> puts it there are two ways of starving a patient (i) simply by depriving him of food (the analogy is diminution of the oxygen content of the blood as in nitrous oxide anæsthesia) and (ii) by giving him abundant

food but simultaneously administering purgatives (the analogy is ether anæsthesia where the oxygen content of the blood is high, but the cells are prevented from using it because they are poisoned by the anæsthetic)

It may well be asked why unconsciousness due to simple oxygen lack which can be so easily produced is not used in surgery. The fact is that in the non resistant type of patient, minor operations *can* be done during unconsciousness produced by this means

To illustrate the possibility of anæsthetising by oxygen deprivation alone we have repeated the old experiment of producing anæsthesia sufficient for dental extraction by nasal administration of nitrogen in 50 cases. Although in all cases unconsciousness was reached in the robust or anæsthetic resistant type the true anæsthetic state was not achieved as indicated by *some response to the stimulus of extraction*. However on the whole anæsthesia was satisfactory and the fact that 'ordinary gas' (i.e. nitrous oxide) was not being used was unknown to dentist and patient

This method of producing unconsciousness is inflexible since it subjects all the tissues of the body to the same degree of oxygen lack. When the surgical stimulus is strong (e.g. in an abdominal operation) the reflex response in the form of muscular rigidity will impede the surgeon unless anæsthesia is profound. If deep anæsthesia is to be produced by oxygen lack alone the blood oxygen has to be reduced to levels which cause grave cardiac embarrassment. Anæsthesia from simple oxygen deprivation is therefore impracticable for other than trivial operations

By the use of anæsthetics a differential reduction of oxygen to the cells of the body is produced. The brain can be subjected to a degree of oxygen lack sufficient to cause unconsciousness at a time when the activity of the other tissues is not seriously impaired

*Whatever the means of entry into the body a drug can be conveyed to the tissues only by the blood stream.* During induction the concentration of the anæsthetic in the blood is high and the factors which determine its distribution to the brain and the other structures are

- 1 The blood supply of the tissues
- 2 Their lipid content

Because the blood supply to the brain through the carotid arteries exceeds that of any other part of the body it follows that the amount of anæsthetic to which it is exposed is proportionately high

DIAGRAM TO ILLUSTRATE BLOOD  
BRAIN AND TISSUE SATURATION  
DURING INDUCTION

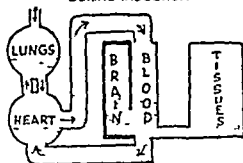


FIG 2 (after Vosworth).—The distribution of blood is the chief factor determining differential distribution of an anæsthetic. Because of the large blood supply of the brain equilibrium between arterial blood and brain is rapidly established during induction.

In the following four diagrams the brain cell (A) is shown as having a larger minute-volume blood supply and greater lipid content than a cell of any other tissue (B). The difference in lipid content of the two cells is indicated by the widths of the cell borders. The depth of shading in the cell border represents the absorption of the anæsthetic by lipids, and the colour of the cell the amount of oxygen available to it. Changes of colour from red to blue indicate progressive diminution of oxygenation and with it of tissue activity.

### Normal Cell Oxygenation

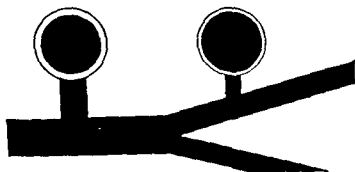


FIG 3

This figure represents arterial blood and cell oxygenation in a normal unanæsthetised subject. The blood carries oxygen in excess of metabolic requirements, and the capacity of the body cells to utilise oxygen is unimpaired.

In the following three diagrams the same depth of anæsthesia is reached by three different methods. The anoxia of the brain cell (A) necessary to achieve this degree of depression is represented by blue

### Effect of any Inert Gas (say Nitrogen) on Tissue Oxidation

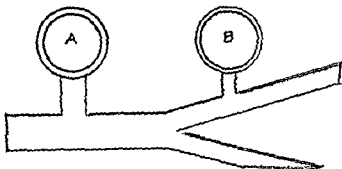


FIG. 4

To attain this degree of anoxia of the brain cell by  $O_2$  deprivation alone (as by the inhalation of an inert gas such as nitrogen) necessitates subjecting all other tissue cells of the body to an equivalent degree of oxygen deprivation. Such a reduction of oxygen to the heart muscle would seriously impair its efficiency. The lipid content of the cells is here immaterial because an anæsthetic is not used.

### Effect of Ether on Tissue Oxidation

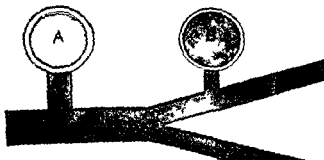


FIG. 5

When ether is used the blood is fully oxygenated and anoxia of the brain cell is achieved by the absorption of the drug by the cell.

lipoid. For reasons already given (less blood supply and less lipoid content of cell) the amount of ether absorbed by the cells of other tissues (B) is less than that absorbed by (A) and the activity of the former is therefore relatively unimpaired. This differential reduction of oxygenation ensures that anaesthesia can be achieved without serious depression of tissues other than the brain.

### Effect of Nitrous Oxide on Tissue Oxidation

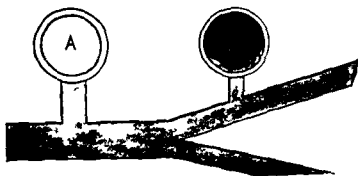


FIG 6

With  $N_2O$  anaesthesia is attained by a combination of the two methods just described and illustrated in figs 4 and 5.  $N_2O$  has definite though weak anaesthetic properties and when absorbed by cell lipoids causes slight reduction in cell oxidation. The additional effect of a reduction of  $O_2$  in the inhaled mixture (and therefore in arterial blood) a factor which affects all cells equally is necessary in order to deepen anaesthesia to the desired level. The reduction of arterial oxygen results in less oxygen being available to other tissues than when ether is being used. This explains why ether is a more suitable anaesthetic than nitrous oxide for cases of myocardial degeneration in which a liberal supply of oxygen to cardiac muscle is essential.

The ratio of the amount of any anaesthetic absorbed by the lipoid-containing cell to the amount which leaves the cell in the venous blood is constant i.e. the fat/water coefficient is constant for any one drug. As administration progresses the absorption of the anaesthetic by other tissues increases until their percentage saturation approaches

In the following three diagrams the same depth of anæsthesia is reached by three different methods. The anoxia of the brain cell (A) necessary to achieve this degree of depression is represented by blue

### Effect of any Inert Gas (say Nitrogen) on Tissue Oxidation

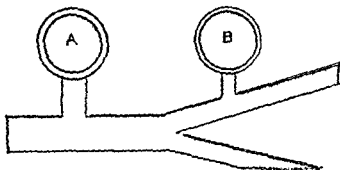


FIG. 4

To attain this degree of anoxia of the brain cell by  $O_2$  deprivation alone (as by the inhalation of an inert gas such as nitrogen) necessitates subjecting all other tissue cells of the body to an equivalent degree of oxygen deprivation. Such a reduction of oxygen to the heart muscle would seriously impair its efficiency. The lipid content of the cells is here immaterial because an anæsthetic is not used.

### Effect of Ether on Tissue Oxidation

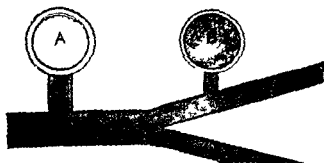


FIG. 5

When ether is used the blood is fully oxygenated and anoxia of the brain cell is achieved by the absorption of the drug by the cell.

lipoid. For reasons already given (less blood supply and less lipoid content of cell) the amount of ether absorbed by the cells of other tissues (B) is less than that absorbed by (A) and the activity of the former is therefore relatively unimpaired. This differential reduction of oxygenation ensures that anæsthesia can be achieved without serious depression of tissues other than the brain.

### Effect of Nitrous Oxide on Tissue Oxidation

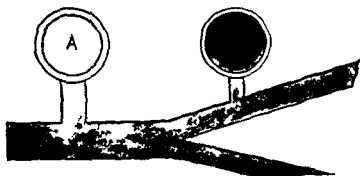


FIG 6

With  $N_2O$ , anæsthesia is attained by a combination of the two methods just described and illustrated in figs 4 and 5.  $N_2O$  has definite though weak anæsthetic properties, and when absorbed by cell lipoids causes slight reduction in cell oxidation. The additional effect of a reduction of  $O_2$  in the inhaled mixture (and therefore in arterial blood) a factor which affects all cells equally is necessary in order to deepen anæsthesia to the desired level. The reduction of arterial oxygen results in less oxygen being available to other tissues than when ether is being used. This explains why ether is a more suitable anæsthetic than nitrous oxide for cases of myocardial degeneration in which a liberal supply of oxygen to cardiac muscle is essential.

The ratio of the amount of any anæsthetic absorbed by the lipoid-containing cell to the amount which leaves the cell in the venous blood is constant, i.e. the fat/water coefficient is constant for any one drug. As administration progresses, the absorption of the anæsthetic by other tissues increases until their percentage saturation approaches



that in the brain. This explains the clinical observation that a small dose intravenously of a barbiturate (say 0.3 g thiopentone) given quickly produces sudden unconsciousness and is followed by quick recovery. A large proportion of it is carried immediately to the brain by the vast carotid circulation the remainder being distributed to the rest of the body. The rapid onset of deep unconsciousness is accounted for by the sudden concentration of the barbiturate in the vascular lipid rich brain tissue. The fat/water (or lipid/plasma) ratio throughout the body must always be adjusting itself to approach the constant. Some 40 to 50 seconds after injection (circulation time of blood = approximately 30 seconds) the arterial blood supplying the brain will contain only a small amount of barbiturate and the lipid/plasma ratio (concentration of anæsthetic in brain lipid/concentration of anæsthetic in venous blood leaving brain) is kept constant by reabsorption into the blood of some of the drug from the brain. Rapid recovery of consciousness is due to the barbiturate being distributed by this means from the brain to other tissues with a less abundant blood supply. If the same amount of barbiturate is given slowly at no time will there be such a high concentration in the brain because the drug is from the first distributed more uniformly throughout the tissues. In fact the amount in the brain may not be enough to produce unconsciousness. In an animal killed soon after a rapidly induced anæsthesia a large proportion of the anæsthetic is found in the brain. This disproportion contrasts with the more uniform distribution of the anæsthetic drug throughout the body tissues after prolonged narcosis.

With any intravenous anæsthetic the duration of anæsthesia does not vary directly with the amount of drug injected. If thiopentone 0.3 g is given and 2 minutes anæsthesia obtained a dose of 0.9 g injected at the same rate to the same patient might well furnish anæsthesia of 20 minutes duration. The explanation is that in the case of the larger dose even after equalisation of the concentration of anæsthetic in the other tissues and the brain the brain concentration is sufficiently high for the patient still to be unconscious. Lightening of anæsthesia can now take place only by excretion of the drug from the body and not as after injection of a small amount by its redistribution from a greater to a lesser potential.

When administration of a volatile anæsthetic ceases the blood concentration is lowered by excretion of the anæsthetic in the expired air. As the tension of anæsthetic in the blood thus falls below that in the brain and other tissues anæsthetic is excreted into the blood that is anæsthetic always travels from a high to

a low tension. In this way redistribution and elimination of any agent is effected.

Nitrous oxide is a weak anæsthetic. As far as potency is concerned, it should be regarded as occupying a position between the inert gases such as nitrogen and an anæsthetic of full potency such as ether. Provided that adequate oxygen is given to support basal metabolic requirements and that the patient is fit and unpremedicated, it is not possible with nitrous oxide to produce deep unconsciousness. Although nitrous oxide is absorbed by the cell lipoids it is only a weak inhibitor of oxidation. To enhance its feeble action the oxygen content of the arterial blood supplying the cell must be reduced, particularly in the resistant patient. If a robust patient remained anæsthetised with a mixture of  $N_2O$  and 10 per cent  $O_2$  the raising of the  $O_2$  percentage to 13 might lighten anæsthesia to such an extent as to make the patient unmanageable. The lightening of anæsthesia is due not to his being given 3 per cent less  $N_2O$  but to his being given 3 per cent more  $O_2$ .

Some authorities hold that nitrous oxide has no anæsthetic effect, but acts only as an oxygen replacer. If this were so any inert gas would furnish as satisfactory an anæsthesia as does nitrous oxide. If nitrogen is administered to a robust patient for dental extraction it becomes apparent that its anæsthetic properties are not as effective as are those of nitrous oxide. It is possible to induce anæsthesia in an anæmic patient with nitrous oxide and 20 per cent oxygen. This would be impossible if nitrous oxide were simply an oxygen replacer, since the same patient is fully awake when breathing a mixture of nitrogen and 20 per cent oxygen—that is atmospheric air. Nitrous oxide should be regarded as a mild inhibitor of one of the enzymes in the process of cell oxidation.

Although the many different anæsthetics all produce unconsciousness the clinical pictures resulting from their use show minor variations. Fats differ in structure and it is probable that lipoids in different parts of the brain have selective affinities for particular anæsthetics. This would explain why some anæsthetics (e.g. thiopentone) in producing a given depth of anæsthesia depress the respiratory centre more than others. Generally speaking the safety of an anæsthetic drug decreases in proportion to the involvement of the respiratory centre in the general depression of the nervous system.

Of the various theories each having something to commend it, the most reasonable and that which best explains the action of every known type of anæsthetic is the oxygen deprivation theory. Incontrovertible evidence may some day be produced to account for the

phenomenon of anæsthesia and it is possible that it will confirm this theory. This working hypothesis at least has the merit of allowing a student to learn how to achieve with safety the type of anæsthesia required in any particular case.

#### REFERENCE

- (1) Macklin A. H. 1931 *Lancet* 2, 897

## CHAPTER IV

### RESPIRATION

WE breathe for two reasons—to obtain oxygen for the tissues and to exhale carbon dioxide an end product of tissue metabolism. These two functions are equally necessary for the maintenance of life. The essential importance of oxygen is appreciated by every human. Normally, carbon dioxide is a respiratory stimulant, but in excessive quantities it becomes a tissue poison and respiratory depressant. Except by misuse of the rebreathing bag of an anæsthetic apparatus or by deliberate experiment the condition of  $\text{CO}_2$  excess in the body does not arise unless oxygen-lack is also present. For instance respiratory obstruction prevents not only the excretion of  $\text{CO}_2$  but also the entrance of oxygen into the body. The patient dies of asphyxia, and though the effect of oxygen-lack may be augmented by  $\text{CO}_2$  excess, the former is probably the main cause of death.

### THE MECHANISM OF RESPIRATION

Breathing is a muscular act just as much as is walking. In breathing the lungs themselves do no work in drawing in air. In expiration too they are passive except for the recoil of their elastic tissue stretched during inspiration. Muscular action causes alternate expansion and contraction of the thoracic cavity by which means air is drawn into and expelled from the lungs. (When the volume of the thoracic cavity is increased the pressure inside the thorax is reduced and the difference between pressures inside and outside the chest causes a movement of air into the lungs.)

#### The Muscles of Respiration

1 The diaphragm—The most important respiratory muscle is the diaphragm. This large umbrella shaped, fibromuscular septum separates the thorax from the abdomen. Its convex surface forms the floor of the thorax its concavity the roof of the abdominal cavity. The peripheral part of the diaphragm is muscular the centre tendinous. During inspiration the muscle of the diaphragm contracts its dome

phenomenon of anæsthesia and it is possible that it will confirm this theory. This working hypothesis at least has the merit of allowing a student to learn how to achieve with safety the type of anæsthesia required in any particular case.

#### REFERENCE

- (1) Macklin A H 1931 *Lancet* 2, 897

thoracic cavity is thus markedly increased, if, however, there is any obstruction to respiration, the volume of the thoracic cavity is not greatly altered but the negative intrathoracic pressure increases considerably

Forced expiration is assisted by powerful contraction of the abdominal muscles

### Significance of Activity of the Accessory Muscles of Respiration

Except in violent exercise when increased respiratory exchange is essential it is improbable that the reader will often see the accessory inspiratory muscles functioning as respiratory aids. If the patient is at rest the accessory muscles of respiration come into play only if there is respiratory obstruction, and then only if he is conscious. The aid of these muscles is essential to the attempt to overcome the effect of respiratory obstruction and if the anaesthetist by producing unconsciousness removes the action of these voluntary muscles, he may be responsible for a preventable death. The management of anaesthesia in a patient suffering from respiratory obstruction from oedema of the larynx is dealt with on page 303

### THE VOLUME OF THE RESPIRED AIR

The maximum capacity of the lungs is about 5 litres. The greatest volume of air which can be exhaled following the deepest possible inhalation is known as the vital capacity, and is about 4 litres. The volume of the residual air i.e. the air which still remains in the lungs is thus approximately 1 litre. During quiet breathing the lungs contain about 2.5 litres and the amount of air entering and leaving the lungs during respiration is about 400 c.c. This latter is known as the tidal air, of which the amount remaining in the nasopharynx, trachea and bronchi approximately 150 c.c. is known as the dead-space air because it takes no part in the gaseous exchange. The volume of the tidal air is variable whereas that of the dead space air for all practical purposes remains constant.

Effective respiration is the difference between the tidal air and the dead-space air. In an average quiet respiration it is therefore  $400 \text{ c.c.} - 150 \text{ c.c.} = 250 \text{ c.c.}$  Calculation of effective respiration shows that one deep breath of 600 c.c. ( $600 - 150 = 450$ ) introduces as much oxygen as nine shallow breaths of 200 c.c. ( $200 - 150 = 50$ ,  $50 \times 9 = 450$ ). To appreciate this is to realise the ineffectiveness of

becomes flattened pushing down the abdominal viscera. In expiration it relaxes and the volume of the thoracic cavity is diminished as the diaphragm is pushed upwards by the abdominal viscera. This action is normally assisted by the contraction of the abdominal muscles.

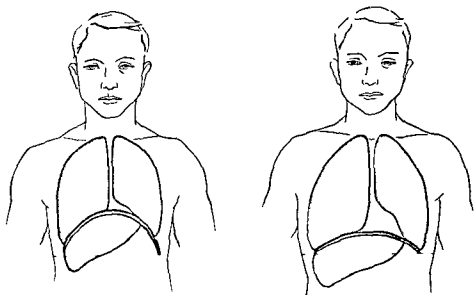


FIG 7

- (L.) *Expiration*—The relaxed diaphragm is forced upwards by contraction of the abdominal muscles on the abdominal viscera.  
 (R.) *Inspiration*—The dome of the diaphragm is flattened by contraction. The diameter of the thorax is increased.

**2 Muscles raising and depressing the ribs in quiet respiration**—In quiet inspiration the external intercostal muscles raise the ribs and so increase the volume of the thorax.

In expiration the contraction of the internal intercostals depresses the ribs. The external and internal intercostals are assisted in their respective functions by muscles of less importance.

**3 Accessory muscles of respiration**—In emergency conditions necessitating forced inspiration the full action of the inspiratory muscles already described is augmented by others which include the sterno mastoid pectorales and trapezius. These muscles of the neck and shoulder are attached below to the upper part of the thorax and when they contract they raise the sternum and upper ribs upwards and outwards. Normally in forced inspiration the volume of the

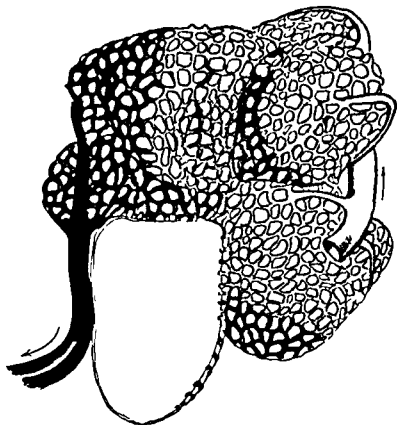


FIG 8 (After Miller 1893) —Alveoli of the lung are shown covered with blood capillaries. An alveolus is seen cut across Greatly magnified

It is estimated that owing to these divisions, the surface or breathing area of the lungs is 200 square metres<sup>2</sup> or 110 times that of the skin covering the body On the assumption that the 2 500 c c of air in the moderately distended lungs is spread equally over this area the thickness of the resultant layer of air is of the order of 0.01 mm. The exchange of gases between the air inside the alveolar sacs and the blood in the capillaries surrounding them is facilitated because the walls of both these structures consist of only one layer of flattened endothelial cells. The ultimate air containing saccules of the lungs are called alveoli and the air they contain is referred to as alveolar air.

Any anaesthetic to be inhaled must be in the form of a gas. The transference from the lungs to the tissues of a gas (e.g. nitrous oxide or ether vapour) to maintain anaesthesia and of oxygen to maintain life and the transference in the reverse direction of carbon dioxide involve processes easy to understand. Gases separated by a membrane



shallow breathing and that one breath of oxygen has less influence in diluting the average gaseous contents of the lungs (2 500 c c ) than is commonly imagined

## PHYSICAL CONSIDERATIONS

The transfer of gases from the lungs to the blood and in the reverse direction is governed by the gas laws Dalton's Law (1807) states that if two gases which do not react together chemically are mixed the pressure exerted by the mixture is equal to the sum of the pressures of the component gases. In a mixture the gases should be thought of individually and the share which each one has in the total pressure is called its partial pressure. This pressure is proportional to the concentration of that gas in the mixture. In a closed vessel containing a mixture of 4 parts of nitrogen and 1 part of oxygen the pressure exerted by the oxygen is the same as if the one part of oxygen were enclosed in the vessel alone. The same applies to the nitrogen.

The quantity of a given gas which a liquid dissolves depends on (i) the solubility of the gas (ii) the pressure of the gas to which the liquid is exposed and (iii) the temperature of the liquid. When equilibrium is attained the tension of the gas in the liquid is the same as the tension of the gas in the atmosphere adjoining the liquid.

## GASEOUS EXCHANGE IN THE LUNGS

The lungs are composed of fibrous muscular and elastic tissues in contact on one side with the alveolar air and covered on the other side with minute blood capillaries. If the interior of the lung were a single hollow chamber like a balloon only a very small proportion of the air within would come into contact with the surface. The lungs are divided and subdivided many times so that the surface of the terminal minute alveolar sacs is very extensive providing a very great area for contact of air with the lung epithelium and its surrounding capillaries. The exchange of gases is thus greatly facilitated.

The composition of alveolar air differs from that of the outside atmosphere in that it has given up some oxygen to, and taken up carbon dioxide from the blood passing through the lungs. Expired air is a mixture of alveolar air and atmospheric air from the dead space

✓

	Inspired Air (atmospheric)	Alveolar Air	Expired Air
O <sub>2</sub>	20.96 per cent	14 per cent	16 per cent
CO <sub>2</sub>	0.04 per cent	5.5 per cent	4.4 per cent
N <sub>2</sub> *	79 per cent	80.5 per cent	79.6 per cent
Water vapour	Unsaturated in varying degree	Saturated	Practically saturated

Certain apparent discrepancies will be noticed in the table. If all the oxygen absorbed were used by the body to oxidise carbon only a volume of CO<sub>2</sub> equal to the volume of O<sub>2</sub> used would be expired and the sum of the volumes of O<sub>2</sub> and CO<sub>2</sub> in inspired air, alveolar air, and expired air would be the same. Actually a little of the oxygen is used to oxidise hydrogen from food to H<sub>2</sub>O and since water vapour is not taken into account in the table the volume of *dry* gases expired is somewhat less than the volume inspired. As the volume of nitrogen remains unchanged its percentage in the expired air will be greater than in the inspired

The pressure of gases within the lungs is approximately the same as that of the outside atmosphere (760 mm Hg). Since the alveolar air is saturated with water vapour which exerts a partial pressure of 45 mm Hg, the sum of the partial pressures of the remaining gases is  $(760 - 45 = 715)$  mm Hg. The tension of oxygen in the alveolar air is therefore  $14/100 \times 715 = 100$  and that of carbon dioxide is  $5.5/100 \times 715 = 40$

The oxygen requirement of a normal person at rest is such that about one third of the oxygen content of the arterial blood is removed during its circulation through the tissues. An average sample of

\* Including other inert gases which total approximately 1 per cent

freely permeable to them tend to come into equilibrium so that the pressure exerted by (or tension of) a gas becomes identical on both sides of the membrane. An everyday illustration of this is provided by the bottle of soda water which contains  $\text{CO}_2$  bottled under tension. When the stopper is removed, the  $\text{CO}_2$  bubbles off until its tension in the water is equal to that in the surrounding air. If on removal of the stopper the soda water were separated from the air by a permeable membrane the process would still take place.

### The Exchange of Oxygen and Carbon Dioxide

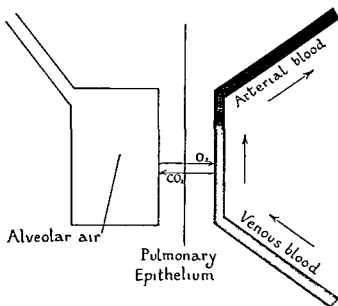


FIG 9

Alveolar air and arterial blood are in equilibrium so far as the tensions of oxygen (100 mm Hg) and of carbon dioxide (40 mm Hg) are concerned. Venous blood changes to arterial blood in the lungs by giving up carbon dioxide to and taking in oxygen from the alveolar air through the pulmonary epithelium.

forcing the gas from the alveoli to the blood is very high, and induction is therefore rapid. After removal of the mask, the pressure gradient will be in the reverse direction. The factors which determine the speed of elimination of a gaseous anæsthetic are the rate with which the tissues give it up (rapid in the case of nitrous oxide) and the difference in pressure (this time in the opposite direction) between the nitrous oxide in the blood and that in the pulmonary alveoli. As this is also great, recovery from nitrous oxide will be rapid. With ether, induction is inevitably slower. One reason is that because of its irritating properties, a high concentration of the vapour cannot be breathed at the commencement with the result that in the early stages the partial pressure of ether in the alveoli remains small. Recovery also is slower than from nitrous oxide, because, after administration is stopped, the tissues give up ether slowly to the blood and therefore the pressure gradient from the blood to the lungs must necessarily be small.

## TRANSPORT OF GASES BY THE BLOOD

### Oxygen

Oxygen is transported in the blood by the hæmoglobin in the red blood corpuscles. The hæmoglobin of arterial blood is normally 95 per cent saturated with oxygen. The amount of oxygen with which hæmoglobin combines depends on the oxygen tension to which it is exposed. Under normal conditions (in alveolar oxygen tension of approximately 100 mm Hg) the amount is maximal and is 1.34 c.c. of oxygen per 1 gm. of hæmoglobin. Although exposure to a higher tension of oxygen (e.g. by inhalation of pure oxygen) increases the amount dissolved in the blood plasma, it cannot increase, to any appreciable extent, the amount taken up by the hæmoglobin since this is already almost saturated with oxygen.

Although an increase of oxygen tension above the normal 100 mm Hg in alveolar air cannot increase the amount of oxygen taken up by the hæmoglobin, a decrease in tension is followed by a decrease in percentage saturation of hæmoglobin with oxygen. The percentage saturation of hæmoglobin with oxygen is not in direct proportion to the oxygen tension to which it is exposed. The actual relation is expressed by the well-known association curve of hæmoglobin.

venous blood returning to the heart shows 100 c.c. to contain about 13.5 c.c. of oxygen at a pressure of 35 mm Hg

The oxygen tension of alveolar air (100 mm Hg) is greater than the oxygen tension of venous blood (35 mm Hg). There is an effective pressure of  $(100 - 35)$  mm Hg driving oxygen from the alveolar air into the blood. This difference of 65 mm Hg is known as the pressure gradient. The process of raising the partial pressure of oxygen in the venous blood from 35 to 100 mm Hg is part of the transformation of venous blood into arterial blood. This pressure gradient is sufficient to cause the hemoglobin, which in the average venous blood is only two thirds saturated with oxygen, to become almost completely saturated by taking up an additional 7 c.c. of oxygen per 100 c.c. of blood, so that 100 c.c. of arterial blood contain 21.5 c.c. of oxygen at a pressure of 100 mm Hg. The arterial blood gives up oxygen to the tissues according to their metabolic requirements. The blood leaving the tissues with its oxygen tension considerably diminished returns to the lungs as venous blood and so the cycle goes on.

The partial pressure in mm Hg of carbon dioxide in the tissues varies between 50 and 70. In venous blood it is 45 and in alveolar air 40. These figures show that there is always a pressure gradient of carbon dioxide from the tissues, via the venous blood to the alveolar air. The pressure gradient driving carbon dioxide in the reverse direction to oxygen across the pulmonary epithelium i.e. from venous blood into the alveolar air is only 5 mm Hg  $(45 - 40 = 5)$ . This is a much smaller pressure gradient than that for oxygen, but since carbon dioxide is highly diffusible through the alveolar membrane the gradient is sufficient, even during the short time taken by the blood to traverse the pulmonary capillaries to allow equilibrium to be established between the venous blood and the alveolar air. Carbonic anhydrase, an enzyme in the red corpuscles, accelerates the liberation of carbon dioxide from the blood. In passing through the lung capillaries every 100 c.c. of blood transfers approximately 6 c.c. of  $\text{CO}_2$  to the alveolar air.

### The Exchanges of Anæsthetic Gases and Vapours

During induction of inhalation anæsthesia the tension of the anæsthetic gas is higher in the alveoli than in the blood. One factor which influences the rapidity of induction is the magnitude of this pressure gradient (gas tension in alveoli minus gas tension in blood). When undiluted nitrous oxide is inhaled the pressure gradient

anæmia Thus in a patient with 50 per cent hæmoglobin the blood can carry only half as much oxygen as can the blood of a normal person with 100 per cent hæmoglobin

(ii) The serious effect of even a small reduction of alveolar oxygen tension on patients suffering from anæmia is considered on pp 57 and 74

### Nitrous Oxide

In common with other anæsthetics nitrous oxide is carried partly within the red cells and partly in solution in the blood plasma During nitrous-oxide anæsthesia the oxygen content of the inspired mixture is low The alveolar oxygen tension is diminished, with corresponding diminution of the percentage saturation by oxygen of the circulating hæmoglobin Should cyanosis exist it is due to the presence of reduced hæmoglobin (i.e. hæmoglobin not combined with oxygen) and in no way is it specifically associated with the presence of nitrous oxide in the blood

### Carbon Dioxide

Blood can take up about twenty times the amount of carbon dioxide that water can at the same temperature and under the same carbon dioxide pressure Thus about 5 per cent of the  $\text{CO}_2$  carried in the blood is in solution, the remaining 95 per cent being in chemical combination, mainly in the form of bicarbonate ions In order to explain the transport of  $\text{CO}_2$  reference will first be made to certain general physico chemical principles, afterwards their application to blood will be discussed

## PHYSICO CHEMICAL PRINCIPLES \*

Any aqueous solution contains electrically charged particles known as ions Pure water is composed very largely of undissociated molecules of  $\text{H}_2\text{O}$  but some molecules are dissociated to form hydrogen ions ( $\text{H}^+$  acid) and hydroxyl ions ( $\text{OH}^-$  alkaline) The  $\text{H}$  ions are charged with positive and the  $\text{OH}$  ions with negative electricity Every aqueous solution contains both  $\text{H}$  and  $\text{OH}$  ions and the product of their concentrations, measured in gram equivalents per litre is found to be constant  $10^{-14}$  The reaction (acidity or alkalinity) depends on the ratio of  $\text{H}^+$  to  $\text{OH}^-$

\* We are indebted to S. L. Cowan D.Sc. for assistance with this section

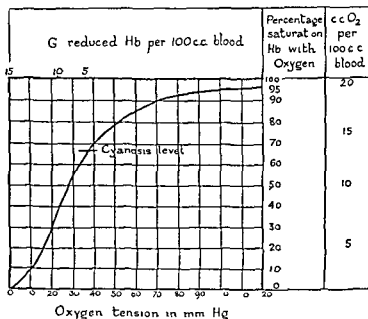


FIG 10

Oxygen association (or Oxygen-dissociation) Curve of Hæmoglobin —  
The curve is that of hæmoglobin in blood at 37° C containing CO<sub>2</sub> at a pressure of 40 mm Hg. Within limits, increase of carbon dioxide assists the unloading of oxygen from the hæmoglobin to the tissues so that association curves will vary with the CO<sub>2</sub> tension in the blood. This factor is not of practical importance to the anæsthetist and the CO<sub>2</sub> tension in the blood should never be increased deliberately with the object of assisting tissue oxidation.

The effect on blood oxygenation and on the colour of a normal patient of inhaling mixtures containing various percentages of oxygen is shown in fig 12 p 55

The total amount of oxygen which arterial blood carries depends on two factors. These are (i) the hæmoglobin content of the patient's blood and (ii) the oxygen tension to which it is exposed.

(i) Each 100 c.c. of blood of an average person contains 15 g. of hæmoglobin. Since each 1 g. combines with 1.34 c.c. of oxygen, it follows that 100 c.c. of blood can carry, by means of its hæmoglobin,  $15 \times 1.34 \text{ c.c.} = 20 \text{ c.c.}$  of oxygen. Besides this, a small amount about 0.3 c.c. is carried in solution in the plasma. The oxygen carrying capacity of the blood is proportionately reduced by any degree of

anæmia Thus in a patient with 50 per cent hæmoglobin the blood can carry only half as much oxygen as can the blood of a normal person with 100 per cent hæmoglobin

(ii) The serious effect of even a small reduction of alveolar oxygen tension on patients suffering from anæmia is considered on pp 57 and 74

### Nitrous Oxide

In common with other anæsthetics, nitrous oxide is carried partly within the red cells and partly in solution in the blood plasma During nitrous oxide anæsthesia, the oxygen content of the inspired mixture is low The alveolar oxygen tension is diminished with corresponding diminution of the percentage saturation by oxygen of the circulating hæmoglobin Should cyanosis exist it is due to the presence of reduced hæmoglobin (i.e. hæmoglobin not combined with oxygen) and in no way is it specifically associated with the presence of nitrous oxide in the blood

### Carbon Dioxide

Blood can take up about twenty times the amount of carbon dioxide that water can at the same temperature and under the same carbon dioxide pressure Thus about 5 per cent of the  $\text{CO}_2$  carried in the blood is in solution, the remaining 95 per cent being in chemical combination mainly in the form of bicarbonate ions In order to explain the transport of  $\text{CO}_2$  reference will first be made to certain general physico chemical principles afterwards their application to blood will be discussed

## PHYSICO-CHEMICAL PRINCIPLES \*

Any aqueous solution contains electrically charged particles known as ions Pure water is composed very largely of undissociated molecules of  $\text{H}_2\text{O}$  but some molecules are dissociated to form hydrogen ions ( $\text{H}^+$  acid) and hydroxyl ions ( $\text{OH}^-$  alkaline) The  $\text{H}^+$  ions are charged with positive and the  $\text{OH}^-$  ions with negative electricity Every aqueous solution contains both  $\text{H}^+$  and  $\text{OH}^-$  ions and the product of their concentrations, measured in gram equivalents per litre is found to be constant  $10^{-14}$  The reaction (acidity or alkalinity) depends on the ratio of  $\text{H}^+$  to  $\text{OH}^-$

\* We are indebted to S. L. Cowan D.Sc. for assistance with this section



The reaction of a solution is said to be neutral when the H and OH ions are present in equal concentrations ( $10^{-7}$ ). This is the case with pure water in which the formation of an H ion must of necessity be accompanied by that of an OH ion  $\text{H}_2\text{O} \rightleftharpoons \text{H}^+ + \text{OH}^-$ . When the

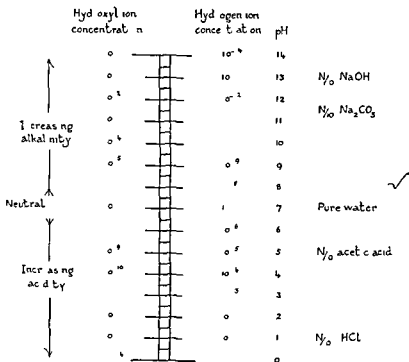


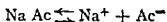
FIG. 11.—This scale includes the whole range of possible degrees of acidity and alkalinity of any aqueous solution. To express the reaction of a solution in terms of OH or of H ion concentration would be cumbersome. To obviate this a pH scale is used. It will be seen that the pH value is the logarithm of the reciprocal of the H ion concentration, or, more simply, it is the index number of the H ion concentration with the negative sign changed to positive.

H ion concentration is the greater the solution is acid and when OH ions are in excess the reaction is alkaline. No matter whether H or OH ions preponderate the reaction of the solution is always expressed in terms of the concentration of hydrogen ions present. In an acid solution this ranges from  $10^{-6}$  to  $10^{-1}$  and in an alkaline solution from  $10^{-1}$  to  $10^{-14}$  gram equivalents per litre. An H ion concentration of 1 g equivalent per litre corresponds to 1 g of hydrogen ions per litre. In a solution with an H ion concentration of  $10^{-3}$  the weight of H ions is  $10^{-3}$  (i.e. 0.001) g per litre. The reaction of an aqueous

solution can be expressed in terms of  $cH$  (H ion concentration), but as it is difficult to form a conception of such small weights it is more convenient to refer to the concentration in terms of a logarithmic scale where the units are simple numbers. Only the numerical value of the power of the H ion concentration is used, omitting the negative sign. The figure is then referred to as the pH. Thus, for an acid solution with an H ion concentration of  $10^{-3}$ , the pH is 3, and this denotes that the solution contains 0.001 g (1 mg) of H ion per litre. An alkaline solution with an H ion concentration of  $10^{-9}$  (or 0.000000001) g per litre is referred to as having a pH of 9. It will be seen that the lower the index figure the greater is the concentration of H ions and the more acid the solution.  $10^{-3}$  (1/1000) being a greater concentration than  $10^{-9}$  (1/1000000000). As already stated, the H ion concentration of a neutral solution one which yields an equal number of H and OH ions as does pure water is found to be  $10^{-7}$ . The pH of such a solution is 7. The pH of an acid solution lies between 0 and 7 and that of an alkaline solution between 7 and 14. A strong acid e.g. hydrochloric acid, is one which in solution behaves as if it were dissociated almost completely into its constituent ions yielding a high concentration of H ions and having therefore a low pH. A weak acid such as lactic acid dissociates less, the number of free H ions is less and the pH is therefore higher.

Any solution containing the salt of a weak acid has the property of preventing a change in pH when a stronger acid is added to it, until a critical point is reached. The salt of a weak acid is said to act as a *buffer* i.e. it damps down changes in pH which would otherwise occur when a stronger acid is added. The quantity of strong acid which can be added to the solution before any appreciable change of pH occurs is a measure of the *buffering power* of that solution. Salts of weak bases, amino acids or proteins may also act as buffers.

The salt of a weak acid and a strong base, e.g. sodium acetate, when dissolved in water is dissociated partly into its constituent positive and negative ions



In any aqueous solution the existence of a high concentration of acetate ions is impossible since a high proportion of these immediately unite with H ions from the water to form undissociated acetic acid. This leaves Na ions together with the OH ions from the water in dilute solution and since these do not associate to form unionised NaOH the preponderance of OH over H ions results in the solution being alkaline.

The reaction of a solution is said to be neutral when the H and OH ions are present in equal concentrations ( $10^{-7}$ ). This is the case with pure water in which the formation of an H ion must of necessity be accompanied by that of an OH ion  $\text{H}_2\text{O} \rightleftharpoons \text{H}^+ + \text{OH}^-$ . When the

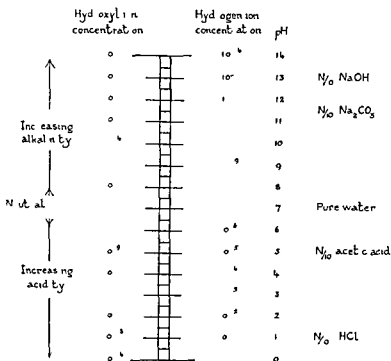
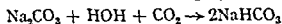


FIG. 11—This scale includes the whole range of possible degrees of acidity and alkalinity of any aqueous solution. To express the reaction of a solution in terms of OH or of H ion concentration would be cumbersome. To obviate this a pH scale is used. It will be seen that the pH value is the logarithm of the reciprocal of the H ion concentration, or, more simply, it is the index number of the H ion concentration with the negative sign changed to positive.

H ion concentration is the greater the solution is acid and when OH ions are in excess the reaction is alkaline. No matter whether H or OH ions preponderate the reaction of the solution is always expressed in terms of the concentration of hydrogen ions present. In an acid solution this ranges from  $10^{-1}$  to  $10^{-7}$  and in an alkaline solution from  $10^{-7}$  to  $10^{-14}$  gram equivalents per litre. An H ion concentration of 1 g equivalent per litre corresponds to 1 g of hydrogen ions per litre. In a solution with an H ion concentration of  $10^{-3}$  the weight of H ions is  $10^{-3}$  (i.e. 0.001) g per litre. The reaction of an aqueous

form hydrogen ions and bicarbonate ions ( $\text{HCO}_3$ ). Of the bicarbonate ions, in their turn a still smaller fraction is ionised to hydrogen ions and carbonate ions ( $\text{CO}_3$ ). The hydrogen ions added in this way are the equivalent of the hydrogen ions of the strong acid ( $\text{HCl}$ ) in the sodium acetate example. The added hydrogen ions can be regarded as combining with the carbonate ions formed by the dissociation of the sodium carbonate to form ions of the stronger acid, first-stage dissociated carbonic acid. This mopping up of the added H ions continues until all the sodium carbonate has dissociated and its carbonate ions have become bicarbonate ions.

The main chemical changes resulting from the addition of  $\text{CO}_2$  to plasma can be summarised in the following equation



Through the transformation of sodium carbonate, the salt of a weak acid into sodium bicarbonate the salt of a stronger acid the solution has been enabled to absorb a considerable amount of  $\text{CO}_2$  with extremely little change of pH.

### THE REACTION OF THE BLOOD AND THE TRANSPORT OF CARBON DIOXIDE\*

By reaction of the blood is meant the degree of acidity or alkalinity of the plasma. This is expressed in terms of pH. The reaction of the blood remains almost constant the limits of variation being pH 7.3 and pH 7.6. Reactions outside these narrow limits cause cell death and are therefore lethal to the organism. When one considers that acid products resulting from metabolic activity are constantly being added to the blood-stream it is a striking fact that the reaction of the blood varies so little. The capacity of the blood to prevent changes in its reaction which would be incompatible with life is well illustrated by comparing the effect of addition of acid to water with the effect of addition of acid to blood. The addition of 0.1 c.c. of N/10 hydrochloric acid to 10 c.c. of blood (with, say a pH of 7.6) does not reduce the pH below 7.3 whereas a similar quantity of acid added to 10 c.c. of distilled water has a profound effect on the pH—causing it to fall from about 7 to 3.

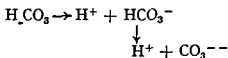
Besides its buffering power blood has two other mechanisms of defence to maintain the constancy of its reaction. The first of these is the excretion of carbon dioxide by the lungs the second is the excretion of any excess of acid or alkali by the kidneys. Further

\* See footnote p. 39

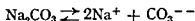
The addition of a stronger acid (i.e. one more dissociated than acetic acid) may be regarded as the addition of  $H^+$  ions. If  $HCl$  is added to a solution of sodium acetate the  $H^+$  ions immediately combine with acetate ions. Since the product of the concentration of the  $H^+$  and  $Ac^-$  ions in the solution always equals a constant fraction of the undissociated acetic acid, more sodium acetate dissociates and this process goes on until practically all the added  $H^+$  ions have united with acetate to form undissociated acetic acid. The ability of the solution to act as a buffer by mopping up  $H^+$  ions persists until the supply of undissociated sodium acetate has been used up. After this point has been reached the addition of even small amounts of strong acid will cause the pH to fall rapidly.

For simplicity in the above explanation the salt of a monobasic acid was chosen. However, in blood plasma the main buffer is sodium carbonate—the salt of a dibasic acid.

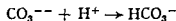
In solution carbonic acid dissociates in two stages, thus



The first stage is the dissociation of a moderately strong acid and the second stage the dissociation of a weaker acid. In plasma the sodium carbonate, a salt of the weaker acid (and the analogue of sodium acetate in the example given) may be regarded as partly ionised into sodium ions and carbonate ions



Since carbonic acid dissociated to the second stage is a weak acid a high concentration of carbonate ions in solution is impossible. Most of these ions immediately join with hydrogen ions from the water to form ions of the stronger acid, first stage dissociated carbonic acid



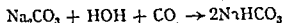
As a consequence of these changes the solution contains mainly the following ions



and it has an alkaline reaction because hydroxyl ions predominate. When the plasma is exposed to a certain tension of carbon dioxide part of the carbon dioxide dissolves. Of the amount dissolved a small and constant fraction is present in the form of undissociated carbonic acid ( $H_2CO_3$ ) and of this carbonic acid a small fraction is ionised to

form hydrogen ions and bicarbonate ions ( $\text{HCO}_3$ ). Of the bicarbonate ions, in their turn, a still smaller fraction is ionised to hydrogen ions and carbonate ions ( $\text{CO}_3$ ). The hydrogen ions added in this way are the equivalent of the hydrogen ions of the strong acid ( $\text{HCl}$ ) in the sodium acetate example. The added hydrogen ions can be regarded as combining with the carbonate ions, formed by the dissociation of the sodium carbonate to form ions of the stronger acid first-stage dissociated carbonic acid. This mopping up of the added H ions continues until all the sodium carbonate has dissociated and its carbonate ions have become bicarbonate ions.

The main chemical changes resulting from the addition of  $\text{CO}_2$  to plasma can be summarised in the following equation



Through the transformation of sodium carbonate the salt of a weak acid into sodium bicarbonate, the salt of a stronger acid, the solution has been enabled to absorb a considerable amount of  $\text{CO}_2$  with extremely little change of pH.

## THE REACTION OF THE BLOOD AND THE TRANSPORT OF CARBON DIOXIDE\*

By reaction of the blood is meant the degree of acidity or alkalinity of the plasma. This is expressed in terms of pH. The reaction of the blood remains almost constant, the limits of variation being pH 7.3 and pH 7.6. Reactions outside these narrow limits cause cell death and are therefore lethal to the organism. When one considers that acid products resulting from metabolic activity are constantly being added to the blood-stream it is a striking fact that the reaction of the blood varies so little. The capacity of the blood to prevent changes in its reaction which would be incompatible with life is well illustrated by comparing the effect of addition of acid to water with the effect of addition of acid to blood. The addition of 0.1 c.c. of N/10 hydrochloric acid to 10 c.c. of blood (with say a pH of 7.6) does not reduce the pH below 7.3 whereas a similar quantity of acid added to 10 c.c. of distilled water has a profound effect on the pH—causing it to fall from about 7 to 3.

Besides its buffering power blood has two other mechanisms of defence to maintain the constancy of its reaction. The first of these is the excretion of carbon dioxide by the lungs the second is the excretion of any excess of acid or alkali by the kidneys. Further

\* See footnote p. 39

reference will be made to these mechanisms after the buffering power of blood and carbon dioxide transport have been considered

If plasma on the one hand and blood on the other are exposed to the same tension of  $\text{CO}_2$  the volume of this gas taken up by the plasma is of the order of one half of that taken up by the blood nevertheless the pH of the plasma falls more. In other words plasma which is in contact with erythrocytes is much better buffered than is separated plasma. The process whereby the erythrocytes augment the buffering power is the so called chloride shift

Preliminary to describing the shift itself it is necessary to mention the composition of the erythrocyte and certain properties of hæmoglobin. The main substance within the erythrocyte is the potassium salt of hæmoglobin. Both oxy hæmoglobin and reduced hæmoglobin are amino acids i.e. in the presence of a strong base they behave as weak acids. Since the concentration of potassium hæmoglobinate within the red cell is large it follows that the contents are heavily buffered.

The shift which involves also a property of the red cell membrane operates in the following way. Some of the  $\text{CO}_2$  which enters the blood from the tissues remains in the plasma, and the rest passes into the corpuscles. In both media the  $\text{CO}_2$  combines with water to form carbonic acid which dissociates into H and  $\text{HCO}_3$  ions. Since the corpuscle has much greater buffering power than the plasma the dissociation proceeds more extensively within the corpuscle than outside it. The membrane of the red cell is permeable to anions such as  $\text{HCO}_3^-$  or  $\text{Cl}^-$  but impermeable (or only slightly permeable) to cations such as  $\text{Na}^+$  or  $\text{K}^+$ . Because of the greater concentration of  $\text{HCO}_3$  ions within the corpuscle a few will diffuse out, and since they cannot be accompanied by ions of the opposite charge, the corpuscle will be left with a net positive charge and will tend to attract negative ions from the plasma. Of these chloride ions, from the NaCl of the plasma are most readily available and are the chief species of ion to pass inwards. Bicarbonate ions from the corpuscle are therefore exchanged for chloride ions from the plasma, and the increase in plasma bicarbonate helps to prevent the rise of acidity of the plasma which would otherwise occur when  $\text{CO}_2$  is added to it.

Reduced hæmoglobin is a weaker acid than is oxy hæmoglobin, and therefore mops up H ions more readily than does oxy hæmoglobin. Reduced blood therefore takes up  $\text{CO}$  in the tissues more readily than does oxygenated blood. Conversely when oxy hæmoglobin is formed again in the lungs the  $\text{CO}_2$  is liberated more readily from the blood.

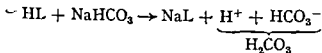
When in the lungs reduced hæmoglobin is transformed to oxy-hæmoglobin, and  $\text{CO}_2$  is excreted into the alveolar air, the chloride shift is reversed

It is known that the combination of carbon dioxide with water to form carbonic acid is a slow reaction, as also is the reverse reaction. Within the erythrocyte is the enzyme carbonic anhydrase, the function of which is to accelerate the reaction in both directions. Without the accelerating action of this enzyme the time which the red cell spends in the tissue capillaries would be too short to enable it to take up the carbon dioxide offered there, and similarly, the time which the red cell spends in the lungs would be too short to enable it to unload the carbon dioxide.

Besides the main buffering systems described above, the proteins of the plasma and the phosphates make a small contribution to the buffering power of blood. Moreover, the  $-\text{NH}_2$  groups of the hæmoglobin have the power of combining very rapidly with about 10 per cent of the carbon dioxide of the blood.

Although blood is a very efficient buffer solution, and is able to absorb considerable quantities of acid or alkali with only a trifling change of pH, its capacity in this respect is not unlimited. The buffer mechanism of the blood is the immediate reaction of the body to any influence tending to displace the pH. If the body did not possess further safety valves for the elimination of excess acid or alkali, either might eventually accumulate to a degree sufficient to displace the pH of the blood outside the limits consistent with life. The safety-valves are the processes of respiration and urinary excretion. The part played by respiration is illustrated by the immediate increase of respiratory exchange which follows moderately vigorous exercise. The  $\text{CO}_2$  resulting from the increased metabolism stimulates the respiratory centre, and respiratory exchange remains above normal until the excess  $\text{CO}_2$  is eliminated.

More vigorous exercise may cause lactic acid, a stronger acid than carbonic acid, to pass into the blood-stream. This results in the production of carbonic acid from the bicarbonate of the plasma



The carbonic acid in its turn becomes dissolved carbon dioxide, respiration is stimulated and the excess carbon dioxide excreted through the lungs. This leaves the subject in a state in which the blood bicarbonate is reduced. A slower readjustment of the pH of the blood



reference will be made to these mechanisms after the buffering power of blood and carbon dioxide transport have been considered

If plasma on the one hand and blood on the other are exposed to the same tension of  $\text{CO}_2$  the volume of this gas taken up by the plasma is of the order of one half of that taken up by the blood nevertheless the pH of the plasma falls more. In other words plasma which is in contact with erythrocytes is much better buffered than is separated plasma. The process whereby the erythrocytes augment the buffering power is the so-called chloride shift

Preliminary to describing the shift itself, it is necessary to mention the composition of the erythrocyte and certain properties of hæmoglobin. The main substance within the erythrocyte is the potassium salt of hæmoglobin. Both oxy-hæmoglobin and reduced hæmoglobin are amino-acids i.e. in the presence of a strong base they behave as weak acids. Since the concentration of potassium hæmoglobinate within the red cell is large it follows that the contents are heavily buffered.

The shift which involves also a property of the red cell membrane operates in the following way. Some of the  $\text{CO}_2$  which enters the blood from the tissues remains in the plasma, and the rest passes into the corpuscles. In both media the  $\text{CO}_2$  combines with water to form carbonic acid which dissociates into H and  $\text{HCO}_3^-$  ions. Since the corpuscle has much greater buffering power than the plasma the dissociation proceeds more extensively within the corpuscle than outside it. The membrane of the red cell is permeable to anions such as  $\text{HCO}_3^-$  or  $\text{Cl}^-$  but impermeable (or only slightly permeable) to cations such as  $\text{Na}^+$  or  $\text{K}^+$ . Because of the greater concentration of  $\text{HCO}_3^-$  ions within the corpuscle a few will diffuse out, and since they cannot be accompanied by ions of the opposite charge, the corpuscle will be left with a net positive charge and will tend to attract negative ions from the plasma. Of these chloride ions, from the  $\text{NaCl}$  of the plasma are most readily available and are the chief species of ion to pass inwards. Bicarbonate ions from the corpuscle are therefore exchanged for chloride ions from the plasma, and the increase in plasma bicarbonate helps to prevent the rise of acidity of the plasma which would otherwise occur when  $\text{CO}_2$  is added to it.

Reduced hæmoglobin is a weaker acid than is oxy hæmoglobin, and therefore mops up H ions more readily than does oxy hæmoglobin. Reduced blood therefore takes up  $\text{CO}_2$  in the tissues more readily than does oxygenated blood. Conversely when oxy hæmoglobin is formed again in the lungs the  $\text{CO}_2$  is liberated more readily from the blood.

The effects of oxygen lack on the respiratory centre can be summarised by stating that a reduction of blood oxygen can affect respiration in two opposite ways. A small reduction causes increased activity of the respiratory centre (via the carotid sinus) with a corresponding increase in ventilation. When the diminution in oxygen intake is considerable, this early indirect stimulating effect is lost and the direct depressant effect operates unopposed, decreasing the sensitivity of the centre to such a degree that the response to an unaltered tension of  $\text{CO}_2$  in the blood is lessened and the volume of pulmonary ventilation is progressively decreased. Oxygen lack must for practical purposes then be regarded as a respiratory depressant. The respiratory centre cannot function properly if it is not supplied with an adequate amount of oxygen and it will quickly cease to function altogether if oxygen deprivation is extreme.

### Depression of the Respiratory Centre

The respiratory centre is rendered less sensitive to carbon dioxide not only by marked oxygen lack but by all drugs used to aid or produce anaesthesia. This is an undesirable but inevitable part of the toxic action of all anaesthetics. Some are more toxic than others in their action on the centre and their use is therefore more likely to result in respiratory depression. Of these morphia is an extreme example. Its action on the respiratory centre is so marked that if given experimentally in doses large enough to produce unconsciousness the respiratory frequency may fall to 10 per minute before this is attained. Respiration remains depressed, and the reduced pulmonary ventilation results in a rise of blood  $\text{CO}_2$  well above that which normally stimulates respiration in fact, only when it reaches this high level will the centre send out an inspiratory stimulus. If administration of morphia were continued until deep anaesthesia resulted the respiratory rate might fall to the dangerously low level of 5 per minute.

A degree of oxygen lack which may not be serious to a lightly anaesthetised patient may be fatal to one whose respiratory centre is already depressed by large doses of anaesthetic drugs. If during light anaesthesia respiration is obstructed for a short time (e.g. by the tongue falling back) it will probably recommence spontaneously as soon as the obstruction is relieved. A similar obstruction in a patient deeply anaesthetised is more serious. Here the effect of oxygen-lack superimposed on a respiratory centre already depressed by anaesthetic drugs may be fatal unless oxygen is supplied to the patient by artificial respiration. The practical point arising from this is that the deeper

then occurs Any lactic acid which is not utilised in the body is excreted through the kidneys in combination with weak bases, rendering the urine acid

## REGULATION OF RESPIRATION

It is evident that a great number of muscle groups are involved in respiration. In forced respiration the nares dilate and contract the vocal cords are actively abducted and in fact, most of the muscles of the abdomen thorax and neck are brought into action. The co ordination of their working is ensured by centralisation of the control of respiration in one part of the brain. This respiratory centre situated in the medulla has an inherent rhythmic activity of its own but it is greatly influenced by outside stimuli and in addition its sensitivity to these stimuli is enhanced or depressed by modifying factors. It is convenient and probably correct to regard carbon dioxide as *the* stimulant to respiration. The respiratory response to a given tension of  $\text{CO}_2$  depends on the state of the respiratory centre at that particular time.

Various conditions render the respiratory centre less or more sensitive to the action of  $\text{CO}_2$ . Of these oxygen lack is the most complicated and will be considered first.

### The Effect of Oxygen-lack

The direct effect of a diminished blood oxygen tension on the respiratory centre is to reduce its sensitivity. This would depress respiration were it not for a compensatory mechanism. The respiratory centre is linked by nervous pathways to the carotid sinus and aortic arch. A moderate diminution of oxygen tension in the blood stream initiates impulses in the carotid sinus and aortic arch which are transmitted by nerves to the respiratory centre rendering it more sensitive. If the oxygen lack is not too severe, this indirect stimulation prevails over the direct depression with a resultant increase in the respiratory rate such as is seen for example in the hurried respirations of deep nitrous oxide anæsthesia. This effect is not seen if the respiratory centre is markedly depressed (e.g. by morphia) in any case it is evanescent and should not be regarded as of practical importance in anæsthesia. It is wrong ever to reduce the oxygen intake in the hope of stimulating respiration. If the oxygen tension is decreased beyond a moderate degree the compensatory action of the carotid sinus reflex is lost and the respiratory centre becomes greatly depressed through the direct effect of oxygen lack.

and covering the mouth until anaesthesia has been deepened sufficiently. If the rare condition of complete nasal obstruction is excepted all patients nose breathe if anaesthetised sufficiently deeply. Mouth breathing is always a sign of light anaesthesia

### (Role of Carbon Dioxide

As has been stated,  $\text{CO}_2$  is the stimulus to respiration. The blood in the lung capillaries and the atmosphere in the lung alveoli are in gaseous equilibrium. Any change in the tension of the gases in the pulmonary capillaries is therefore immediately reflected in a change in composition of the alveolar air and vice versa. During exercise  $\text{CO}_2$  is liberated from the tissues into the venous blood causing increased tension of  $\text{CO}_2$  in the alveolar air and therefore in the arterial blood resulting in stimulation of the respiratory centre. The increased pulmonary ventilation leads to increased loss of  $\text{CO}_2$  by way of the expired air until its tension in the alveolar air is again normal.

It is found experimentally that  $\text{CO}_2$  depletion following prolonged voluntary deep breathing allows the breath to be held longer than would otherwise be possible. The apnoea persists until as the result of metabolic activity the  $\text{CO}_2$  tension in the alveolar air and arterial blood rises sufficiently to stimulate the respiratory centre to such an extent that the subject is forced to breathe again.

During induction of nitrous-oxide anaesthesia in a nervous unpremedicated patient it is often found that after the rapid puffing respiration of excitement a period of apnoea follows the duration of which depends on the degree of hyperpnoea preceding it. This is due to reduction of the  $\text{CO}_2$  content of the alveolar air and blood by the excessive breathing. Such a phase of apnoea may exceed 30 seconds. Since reduced oxygen intake is a necessary accompaniment to induction with  $\text{N}_2\text{O}$  the period of apnoea can result in slight cyanosis. This may disturb the anaesthetist if he is not familiar with the condition and the cause. There is no need for alarm because this state of affairs occurs only during light anaesthesia and the rising  $\text{CO}_2$  content of the blood and alveolar air will soon stimulate the respiratory centre to its normal activity. The fact that breathing is nasal when respiration is resumed after a period of such apnoea is accounted for by the deepened anaesthesia.

Respiration is regulated by the action of  $\text{CO}_2$  on the respiratory centre in such a way that the tension of  $\text{CO}_2$  in the alveolar air is maintained at an almost constant level.

the anæsthesia the more imperative becomes the maintenance of a clear airway and an adequate oxygen supply

The sensitivity of the respiratory centre to  $\text{CO}_2$  may be depressed by any stimulus (e.g. a foreign body or strong ether vapour) to the laryngeal entrance. We infer this from the reactions to an attempt to pass an endotracheal tube when anæsthesia is not deep enough particularly if the patient has been premedicated with morphia. Respirations up to now have been normal but as the tube touches the laryngeal entrance the cords may become closely adducted. Depression of the respiratory centre from the laryngeal irritation is expressed in any one of the following three ways: (a) Respirations continue but their volume is greatly diminished. The cords separate only very slightly insufficiently in fact to allow the passage of a tube. The gaseous exchange however is enough to maintain life. (b) Respiration ceases until the effect on the respiratory centre of the rising  $\text{CO}_2$  tension in the blood forces the patient to separate his cords and inspire. Inspiration here is often preceded by an explosive cough i.e. an expiratory effort. (c) The cords remain approximated and respiration suspended until the heart's action becomes irregular from oxygen-want. The tube has to be removed to allow normal respiration to be resumed.

### **Increase in the Sensitivity of the Respiratory Centre**

The respiratory centre is rendered more sensitive to the action of carbon dioxide by pain excitement fear rage and by slight oxygen deficiency. Increase of breathing during emotion or excitement is a phenomenon familiar to all. It occurs during over activity of the sympathetic nervous system and is probably a primitive reflex the effect of which is to fill the lungs with air in preparation for the sudden muscular activity involved in fight or in flight from danger. It manifests itself not only by an increase in the rate and depth of respiration but also in a change from nasal to oral breathing.

During nasal administration of nitrous oxide increase in sensitivity of the respiratory centre is evidenced (i) during induction where from excitement the patient abandons nasal respiration and breathes rapidly through the mouth and (ii) during anæsthesia too light for the operation being performed. For example if dental forceps are forcefully applied a lightly anæsthetised patient breathing tranquilly through the nose may be stimulated to inspire vigorously through the mouth with the result that anæsthesia lightens rapidly. Nose breathing can be restored only by suspending the operation (so removing the afferent stimuli).

## Causes of Anoxia and their Treatment

(A) Respiration of atmospheres deficient in oxygen—The treatment is obvious

(B) Any interference with respiratory exchange

1 *Respiratory obstruction* as by occlusion of the larynx by the tongue or a foreign body or by laryngeal spasm

2 *Depression of the respiratory centre* as by sedative drugs or by lack of oxygen. This decreases pulmonary ventilation by diminishing both rate and amplitude of respirations

3 *Emphysema phthisis and pneumonia* causing diminished area of air/blood contact

These three diminish the respiratory minute-volume

4 *Mucus in the bronchial tree* And rarely,

5 *Pulmonary fibrosis*

4 and 5 cause decreased permeability by purely mechanical means

Any respiratory obstruction must be removed and conditions 3, 4 and 5 necessitate an increase in the oxygen supply above normal in order to increase the alveolar oxygen tension

(C) Anæmia—A given alveolar oxygen tension results in the oxygenation of a definite *proportion* of the hæmoglobin of the blood. Thus at a fixed tension the oxygen-carrying capacity of the blood depends on its total hæmoglobin content. Reference to the oxygen association curve of hæmoglobin (p 38) shows for example, that when the alveolar oxygen tension is 33 mm Hg the hæmoglobin irrespective of the total amount present is 60 per cent saturated with oxygen. While the degree of oxygen lack resulting from this alveolar oxygen tension is scarcely enough to render an average patient unconscious, it could be fatal to a patient suffering from advanced anæmia

In anæmia, the amount of hæmoglobin available for the transport of oxygen is diminished. Even with a normal alveolar oxygen tension the supply of oxygen to the tissues is diminished because of deficient transport. If the alveolar oxygen tension is lowered the hæmoglobin will be only partly saturated with oxygen so that the supply to the tissues already reduced because of the anæmia will be further diminished. The treatment should be prophylactic to ensure an adequate alveolar oxygen tension and if despite this anoxia still exists the only effective treatment is to provide an increase in the oxygen carrying capacity of the blood by blood transfusion

(D) Diminished rate of circulation—Here although the oxygen tension and the amount of hæmoglobin may be normal the slowly circulating blood does not carry oxygen to the tissues quickly enough

By regulating the excretion of  $\text{CO}_2$ , respiration is one of the important mechanisms by which the reaction of the blood is kept constant. Any alteration in the  $\text{CO}_2$  content of the alveolar air or arterial blood causes an almost instantaneous change in the respiration, which helps to maintain the  $\text{CO}_2$  of the alveolar air and the hydrogen ion concentration of the blood at constant levels.

If the tension of  $\text{CO}_2$  in the alveolar air is raised beyond certain limits this gas loses its stimulating effect and becomes a powerful respiratory depressant. This depression can be produced by prolonged inhalation of an atmosphere containing approximately 10–15 per cent  $\text{CO}_2$ . It follows the inhalation of higher concentrations of  $\text{CO}_2$  more rapidly.

### Nervous Stimuli

The respiratory centre is also affected by impulses from the lungs via the vagi. When the lungs are distended to a certain tension impulses travel up the vagi to the respiratory centre cutting short the inspiratory phase whereupon expiration begins. This phenomenon is known as the Hering-Breuer reflex. Similarly emptying of the lungs is followed by vagal impulses which in turn initiate inspiration. These reflexes have the effect of making respiration less deep and more rapid than it would otherwise be.

Probably this action of the vagus is important in artificial respiration. If the Hering Breuer reflex is not lost forceful artificial deflation of the lungs is followed by impulses causing inspiration. Repeated artificial filling and emptying of the lungs may in this way eventually play a part in the re establishment of natural breathing.

The same effect may be seen when  $\text{N}_2\text{O}$  is administered under pressure. The distension of the lungs so produced may institute a weak expiratory effort. If these two effects are evenly balanced apnœa will result until either the pressure of gas administered is lowered or the alveolar  $\text{CO}_2$  rises sufficiently to force a breath.

### ANOXIA

This is the condition of deficient oxygen supply to the *tissues*. Since death from anæsthesia must be regarded as death from anoxia it is well to see at what points the transfer of oxygen from the atmospheric air to the tissues may break down.

## REFERENCES

- (1) Miller, W. S. 1893 *J. Morph.* 8, 165
  - (2) Winton F. R. and Byliss L. L. 1937 *Human Physiology* London 164
  - (3) Peters J. P. and Van Slyke, D. D. 1931 *Quantitative Clinical Chemistry 1 Interpretations* London 577
- Read also
- Cocking T. T. *pH Values* 5th Edition published by British Drug Houses London
- Drinker C. K. 1938 *Carbon Monoxide Asphyxia* London, New York, Toronto
- Henderson Y. 1938 *Adventures in Respiration* London
- La Motte F. L. Kenny W. R. and Reed A. B. 1932 *Hydrogen Ion Concentration and its Practical Application* London



for their needs. This condition of circulatory insufficiency with fall of blood-pressure may occur in cardiac disease or as a result of shock or overdose of anæsthetic drug. If the condition occurs during anæsthesia administration must be stopped immediately and the head should be lowered to increase the circulation to the medulla.

(E) In the tissues themselves—At least one respiratory enzyme or ferment is present in all body cells. The ability of the cells to utilise oxygen depends on the activity of the enzyme the poisoning of which (the classical example is by cyanide) causes abrupt and fatal termination of the oxidative processes throughout the body. A gradual increase in the concentration of anæsthetic within a cell has a corresponding poisoning effect on the enzyme with corresponding diminution of cell oxidation and activity. If the enzymes of the cells of the respiratory centre are poisoned in this way the centre will suffer from anoxia though abundant oxygen may be circulating in the blood. The respiratory centre is thus depressed and the minute volume exchange falls off appreciably. In the extreme case the respiratory centre is so depressed that it does not respond to any stimulus and respiration ceases. The treatment is to stop administration of the anæsthetic immediately. Provided circulation continues and the respiratory centre is not irreparably poisoned artificial respiration will supply oxygen for the activity of the heart and the continued circulation will result in excretion of some of the anæsthetic drug. Progressive diminution of the tension of anæsthetic in the blood will be followed by a shift of anæsthetic from all the other tissues (including the respiratory centre) until a concentration is reached at which the centre will be able to utilise the oxygen recover its activity and again respond automatically to the stimulus of CO. If interference with the metabolism of the respiratory centre has been severe and prolonged it may never recover its activity and even if it does the damage may be so severe that respiration continues for only a few hours. It should be noted that in this fifth type of anoxia the patient has a good colour even *in extremis*. Because of tissue poisoning he is dying of oxygen want in the midst of plenty.

The gravity of a patient's condition increases if any two or more of the causes of anoxia co-exist.

some degree of cyanosis is usually noticeable. Cyanosis is here unavoidable and not of serious import. It exists in the presence of a free airway and since the light anaesthesia of  $N_2O$  does not depress the respiratory centre the  $O_2$  lack can be relieved at will by the addition of oxygen to the mixture or by allowing the patient a few breaths of atmospheric air.

The great majority of patients can become cyanosed while there is still enough oxygen carried in the blood-stream to support the vital functions of the body but the anæmic patient may die of anoxæmia without showing cyanosis. The presence or absence of cyanosis depends on the quantity of reduced hæmoglobin in the blood. Reduced hæmoglobin is purple (in contrast with fully oxygenated hæmoglobin which is bright red) and when present in the capillaries in sufficient concentration imparts a blue colour to the skin, irrespective of the amount of oxyhæmoglobin present. As has been described (p 38) the quantity of reduced hæmoglobin in the blood is determined by the total hæmoglobin content and the tension of oxygen in the alveolar air to which it is exposed. This latter in turn, is dependent upon (a) the oxygen percentage in the mixture of gases inspired (b) the adequacy of the pulmonary ventilation, and (c) the barometric pressure. The last because its fluctuations in practice, are negligible, is unimportant.

% $O_2$ in inhaled mixture	Alveolar $O_2$ tension in mm Hg	Hæmoglobin % saturation	cc $O_2$ per 100 cc blood	Reduced Hæmoglobin g per 100 cc blood	Colour of patient
21	100	95	20	0	
12	35	66	13	5	
8	28	50	10	7.5	

FIG. 12.—Relationship between the oxygen percentage in the mixture of gases inspired and the colour of the normal patient. For example with 8% oxygen in the mixture the alveolar oxygen tension becomes as low as 28 mm Hg the hæmoglobin is then 50% saturated so that 100 cc of blood carries 10 cc of oxygen and contains 7.5 g of reduced hæmoglobin. The extreme right hand column shows the corresponding colour change.

## CHAPTER V

## CYANOSIS

CYANOSIS is evidence of incomplete oxygenation but its absence whether the patient be conscious or unconscious is not an assurance that the circulating hæmoglobin is fully oxygenated. In the healthy subject breathing air cyanosis may be seen in localised areas in which there is a decrease in the rate of flow of blood through the capillaries as the result of exposure to cold or from increased viscosity of the blood from other causes. the slowly circulating blood is then deprived by the tissues of a greater percentage of oxygen than usual. Cyanosis occurs too in a generalised form during inhalation of atmospheres grossly deficient in oxygen. It is seen also in certain pathological conditions even if the patient is breathing air. The commonest of these are (i) changes in the lungs which retard the transference of oxygen from the alveoli to the capillaries and (ii) cardiac failure resulting in stagnation in the systemic capillaries. Cyanosis may also result from the use of drugs of the sulphonamide group. Apart from this last cause its presence denotes the existence in the blood stream of a considerable proportion of hæmoglobin in the reduced form—i.e. not oxygenated.

In this chapter the occurrence of cyanosis during anaesthesia *in an otherwise healthy patient* is considered and the absence of this colour change in the anæmic subject under similar conditions is also explained.

The anaesthetist must appreciate the ætiology and significance of cyanosis if he is to administer an anaesthetic safely. Anaesthetics with the conspicuous exception of nitrous oxide can produce profound anaesthesia even in the presence of unlimited oxygen and indeed should always be administered in an atmosphere containing at least 20 per cent of oxygen. Under these conditions cyanosis is a danger signal indicating a breakdown in the transfer of this oxygen to the lungs the commonest causes being respiratory obstruction or respiratory arrest. If either of these is present it must be relieved at once.

In nitrous oxide anaesthesia conditions are different. This anaesthetic of limited potency has often particularly in dentistry to be administered without the help of premedication. To be effective in an average patient its weak anaesthetic qualities have to be reinforced deliberately by a reduction of the oxygen intake to such a level that

some degree of cyanosis is usually noticeable. Cyanosis is here unavoidable and not of serious import. It exists in the presence of a free airway and since the light anaesthesia of  $N_2O$  does not depress the respiratory centre the  $O_2$  lack can be relieved at will by the addition of oxygen to the mixture or by allowing the patient a few breaths of atmospheric air.

The great majority of patients can become cyanosed while there is still enough oxygen carried in the blood-stream to support the vital functions of the body but the anæmic patient may die of anoxæmia without showing cyanosis. The presence or absence of cyanosis depends on the quantity of reduced hæmoglobin in the blood. Reduced hæmoglobin is purple (in contrast with fully oxygenated hæmoglobin which is bright red), and when present in the capillaries in sufficient concentration imparts a blue colour to the skin irrespective of the amount of oxyhæmoglobin present. As has been described (p 38) the quantity of reduced hæmoglobin in the blood is determined by the total hæmoglobin content and the tension of oxygen in the alveolar air to which it is exposed. This latter, in turn, is dependent upon (a) the oxygen percentage in the mixture of gases inspired, (b) the adequacy of the pulmonary ventilation and (c) the barometric pressure. The last because its fluctuations in practice, are negligible is unimportant.

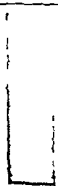
% $O_2$ in inhaled mixture	Alveolar $O_2$ tension in mm Hg	Hæmoglobin % saturation	cc $O_2$ per 100 cc blood	Reduced Hæmoglobin g per 100 cc blood	Colour of patient
21	100	95	20	0	
12	35	66	13	5	
8	28	50	10	7.5	

FIG 12—Relationship between the oxygen percentage in the mixture of gases inspired and the colour of the normal patient. For example with 8% oxygen in the mixture the alveolar oxygen tension becomes as low as 28 mm Hg the hæmoglobin is then 50% saturated so that 100 cc of blood carries 10 cc of oxygen and contains 7.5 g of reduced hæmoglobin. The extreme right hand column shows the corresponding colour change.

Cyanosis will not appear until the alveolar oxygen tension is so far diminished that 5 g (or more) of hæmoglobin per 100 c c of blood remain reduced. The existence of this absolute amount of reduced hæmoglobin in the blood determines the development of cyanosis. This point may be illustrated by comparing the effects of inhalation of a mixture of nitrous oxide with 12 per cent oxygen (which will result in an alveolar oxygen tension of approximately 35 mm Hg) on three types of patients: a man with an average hæmoglobin content (i.e. 15 g per 100 c c of blood), a plethoric man (with, say, 18 g per 100 c c of blood) and an anæmic patient (with say 9 g per 100 c c of blood). Irrespective of whether a person is plethoric, normal or anæmic, an oxygen tension of 35 mm Hg is sufficient to oxygenate only two thirds of the circulating hæmoglobin. The arterial blood of the average man therefore will contain 5 g per cent of reduced hæmoglobin (the capillary blood will contain a higher percentage) and he will be cyanosed. The plethoric man with 6 g per cent of reduced hæmoglobin in his blood will be appreciably cyanosed, while the anæmic patient whose blood will contain only 3 g per cent of hæmoglobin in the reduced form will not be cyanosed at all, since 5 g per 100 c c of blood is the minimum concentration necessary to produce this colour. Calculation of the amount of oxygen carried by the Hb (p 38) shows however that the blood of the plethoric subject despite the fact that he is cyanosed still has a greater oxygen content ( $18 \times \frac{2}{3} \times 1.34 = 16$ ) c c per 100 c c of blood than the normal ( $15 \times \frac{2}{3} \times 1.34 = 13.4$ ) c c and carries double the amount of oxygen ( $9 \times \frac{2}{3} \times 1.34 = 8$ ) c c carried by the blood of the anæmic patient who shows no colour change. Since nitrous oxide is carried in simple solution in the blood plasma, the blood nitrous oxide concentration of these three types of patients will be the same. If the patients are tested for depth of anæsthesia, it will be found that the anæmic is the most profoundly anæsthetised (because he is suffering from the greatest oxygen lack). Even when conscious the diminished hæmoglobin content of his blood subjects him to some degree of  $O_2$ -lack so that his general fitness and with it his resistance to anæsthesia is reduced. The plethoric and the average man are invariably more robust and anæsthetic resistant and so there is greater difficulty in rendering them unconscious and in maintaining anæsthesia.

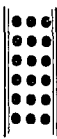
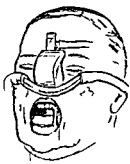
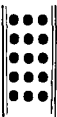
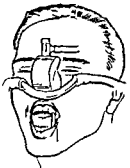


Since a concentration of 5 g of reduced hæmoglobin per 100 c c of blood is necessary to produce cyanosis, it follows that a patient with an advanced anæmia (of say 33 per cent Hb i.e. 5 g of Hb per 100 c c of blood) can never show this colour change. It would be necessary for all his hæmoglobin to be in the reduced form for cyanosis

to occur. Since no oxyhæmoglobin would then be circulating, such a patient must die of oxygen lack before cyanosis could manifest itself.

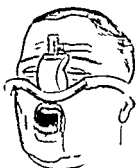
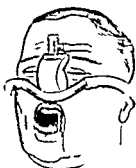
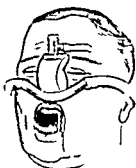
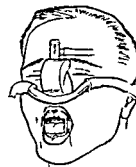
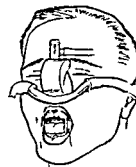
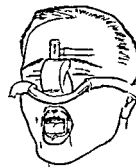



Recognition of the above facts is essential if nitrous oxide is to be given with safety and confidence. In an unpremedicated plethoric man the oxygen content of a nitrous oxide oxygen mixture has to be lowered to such an extent that cyanosis is inevitable if anæsthesia is to be achieved. On the other hand the administration of a similar mixture to an anæmic patient will not cause any change of colour, but, if continued, will produce a truly desperate state of tissue anoxia. If the anæsthetist interprets this latter patient's muscular tranquillity and unchanged colour as proof that anæsthesia is not deep the peace of his fool's paradise will soon be rudely disturbed by the urgent necessity for resorting to artificial respiration.

Fig 13 represents the degree of oxygenation of the blood, and the complexions of the three types of patients described when breathing air, when breathing a mixture of nitrous oxide and 12 per cent oxygen (i.e. sufficient to produce two thirds saturation of the hæmoglobin), and the depths of anæsthesia thereby attained. These latter are indicated by a  $\lambda$  in the last column which depicts the four planes of the third or surgical stage of anæsthesia (fig 15 p 63). The red and blue circles indicate the number of grams of oxyhæmoglobin and of reduced hæmoglobin respectively present in 100 c.c. of blood in these three types of patients.

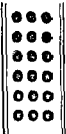
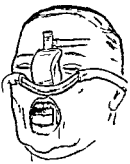

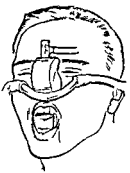

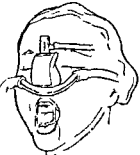
Fig 14 shows the oxygen reduction in a nitrous oxide-oxygen mixture necessary to produce upper first-plane anæsthesia in these patients. In the plethoric patient the oxygen percentage has to be reduced to about 7, and profound cyanosis is inevitable. In the normal, to about 12 which results in mild cyanosis. and in the anæmic only to about 18 and the patient's colour remains unchanged.

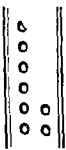
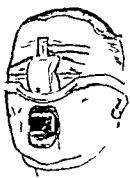
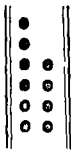
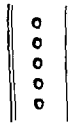

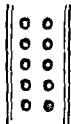


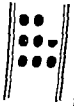
TYPE	G of Hb per 100 cc blood	Hb fully saturated with O <sub>2</sub>	
			APPEARANCE
PLETHORIC	18		
AVERAGE	15		
ANAEMIC	9		

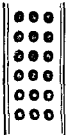
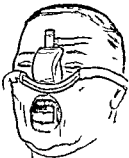
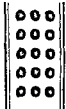
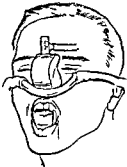

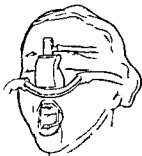
Hb  $\frac{2}{3}$  saturated with  $O_2$   
leaving  $\frac{1}{3}$  as reduced Hb

G of reduced Hb per 100 cc.	APPEARANCE	G of Hb combined $\bar{c} O_2$ per 100cc.	cc of $O_2$ per 100cc blood	Depth of anaesthesia reached	
			12 x 134 = 16	1	
				2	
				3	
				4	
			10 x 134 = 13.4	1	*
				2	
				3	
				4	
			6 x 134 = 8	1	
				2	*
				3	
				4	



TYPE	G of Hb per 100 cc blood	Hb fully saturated with O <sub>2</sub>		Depth of anaesthesia to be reached	
			APPEARANCE		
PLETHORIC	18			1	*
				2	
				3	
				4	
AVERAGE	15			1	*
				2	
				3	
				4	
ANAEMIC	9			1	*
				2	
				3	
				4	

Estimated $O_2\%$ in $N_2O$ $O_2$ mixture	G of reduced Hb per 100cc blood	APPEARANCE	G of Hb combined with $O_2$	cc $O_2$ per 100 cc blood
7				$10 \times 134 = 134$
12				$10 \times 134 = 134$
18				$75 \times 134 = 10$

TYPE	G of Hb per 100 cc blood	Hb fully saturated with O <sub>2</sub>		Depth of anaesthesia to be reached	
			APPEARANCE		
PLETHORIC	18			1	*
				2	
				3	
				4	
AVERAGE	15			1	*
				2	
				3	
				4	
ANAEMIC	9			1	*
				2	
				3	
				4	

With very few exceptions excess of any anæsthetic can bring about death however perfect the airway or abundant the supply of oxygen. To facilitate determination of the depth of anæsthesia it is proposed to employ the very useful plan devised by Guedel<sup>1</sup> of California, of dividing the period between the commencement of induction and the point of death into the four stages (I) analgesia (II) delirium, (III) surgical anæsthesia and (IV) respiratory paralysis shown in the chart (fig 15), and to mark on the chart the changes in the intensity of certain selected nervous activities and reflexes during these stages. Further Guedel subdivided stage III that of surgical anæsthesia, into four planes.

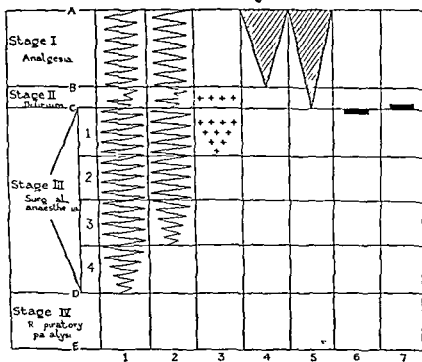


FIG 15 (After Guedel<sup>1</sup>)

Column 1 — Abdominal respiration

2 — Thoracic respiration

3 — Eyeball activity

Column 7 — Vomiting reflex

Column 4 — Eyelash reflex

5 — Eyelid reflex

6 — Swallowing reflex

Guedel based his chart on observations made when administering ether on an open mask to numerous patients. Morphine or other sedatives were not given beforehand sufficient oxygen was always available and needless to say a clear airway was maintained throughout. The conclusions drawn from administering ether roughly hold

## CHAPTER VI

### SIGNS OF ANÆSTHESIA\*

IN considering the effects of administering increasing amounts of an anæsthetic we are concerned primarily with those exerted on the function of the central nervous system as judged by changes in the muscular responses evoked by external stimuli and by changes in respiration. It is only these that are directly observable. There is progressive depression in the function firstly of the cerebral cortex later of the midbrain which is involved in the completion of reflex arcs by which the individual reacts unconsciously to noxious stimuli still later of the spinal cord and lastly of the respiratory centre in the medulla oblongata.

The term unconscious will be used here as implying that the patient cannot give a correct account of what was happening to him or in his immediate environment during that period. A man may be unconscious in this sense even though stimuli are found to provoke reflex actions so complexly co-ordinated that they simulate skilled reactions dependent on paths from and to the cerebral cortex. We assume also that consciousness proper is associated solely with the functioning of the cerebral cortex and that the activities of the rest of the nervous system are purely automatic or reflex.

It is generally easy to tell whether a patient is unconscious or not but in anæsthesia determination of the depth of unconsciousness reached is important and this can be assessed only by the interpretation of landmarks or signs which are frequently present at different stages. Is the patient adequately anæsthetised for the operation contemplated? Is anæsthesia too light and response to stimuli therefore possible unnecessarily deep for the operation being performed or even dangerously deep? We have heard a doctor enquire from time to time during the induction period 'Are you asleep yet?' apparently assuming anæsthesia to be attained when the patient ceased to answer. Other unorthodox procedures not calculated to instil confidence into a patient approaching unconsciousness are to prod his eye vigorously to discover the reaction or to stick a pin into him or pull his hair to see whether there is any response to these stimuli.<sup>1</sup>

\* This chapter deals with anæsthetics other than nitrous oxide. For signs of nitrous oxide anæsthesia see Chapter VII.

does not respond to gentle treatment he must be restrained firmly enough to prevent him damaging himself or others

Struggling during the second stage is liable to occur (i) in the patient who is not really calm but has remained so during the first stage by the exercise of great self-control. During a slow induction the amount of struggling is likely to be proportionate to the nervous tension and the release of mental control dominant during consciousness may result in unexpected violence. Struggling occurs most frequently and most violently in the tract, active type of patient, (ii) if the patient is unpremedicated, (iii) in alcoholics or other drug addicts (iv) when induction is too slow, the patient being kept too long in this stage of potential delirium (v) when the anæsthetic is given badly e.g. as when the concentration of ether vapour is increased too rapidly

The incidence of delirium varies with different anæsthetics, being greatest with inhalation agents particularly if induction is slow. For example induction with ether alone is slow because strong ether vapour is unpleasant, and the patient therefore cannot inhale it in concentrations sufficient to induce anæsthesia rapidly. In contrast to this, when a patient is given an intravenous anæsthetic such as thiopentone the stage of potential delirium is passed through in a matter of seconds. Nitrous oxide also affords a rapid and pleasant induction. Nevertheless struggling occurs not infrequently with this anæsthetic because unless the oxygen intake is markedly diminished it may be impossible to produce surgical anæsthesia in an unpremedicated patient

Delirium may occur also in the second stage *after* an operation when the patient is emerging from the effects of the anæsthetic, though it manifests itself then more often as restlessness than as violent struggling since the patient is back again in bed where noise and external stimuli are less. Further post operative depression decreases any tendency to violent movement. On the other hand there may be sufficient post operative pain to cause restlessness which will persist unless morphia is given

**Third stage**—From level C where stage II passes into stage III muscular depression is sufficient to prevent the patient from moving his limbs in response to stimuli. This level marks the onset of surgical anæsthesia and is recognisable by (i) the onset of automatic respiration, particularly noticeable in cases where the breathing has previously been irregular and (ii) the loss of the eyelid reflex—i.e. closing of the eyes evoked by retraction of the eyelid. Only in the third stage (C-D) that of surgical anæsthesia should surgical operations be performed. As shown in fig. 15 this stage is extensive and is sub-

good for all anæsthetics capable of producing death even when oxygen is abundantly supplied. The chart of course is not invariably accurate for every patient or for every anæsthetic. For this to be possible there would have to be no individual variance in the reactions of patients and all anæsthetics would have to produce identical results. In the chapters on individual anæsthetics we draw attention to signs of anæsthesia characteristic of the particular anæsthetic when these do not conform closely to Guedel's chart.

During the first stage, that of 'analgesia' there is progressive decrease in reaction to painful stimuli and progressive loss of consciousness the latter becoming complete at the end of the stage (line B). If the eyelashes of a conscious patient are touched this mild stimulus will as Human has pointed out evoke movements of the eyelid but this response gradually diminishes until it is lost at the level indicated by line B. The first stage is generally easily recognised. The patient is conscious and generally co-operative. Occasionally there is hysteria which may turn into struggling or delirium in the second stage. In any case it is difficult to draw a sharp line between the end of the first stage and the beginning of the next.

The second stage is known as that of delirium although the delirium may be merely potential or may be manifested by incoherent talk or actual struggling. During this stage the patient is unconscious and will subsequently remember nothing of what has happened. He may become turbulent or remain peaceful according to the anæsthetic used, the type of patient he is, the presence or absence of stimuli, and the skill of the anæsthetist. Conscious control is removed and he may struggle either from a fear the existence of which had been concealed during the first stage or because he is being subjected to some stimulus e.g. pain from premature incision or extraction or even the noise made by dropping instruments or as a response to injudicious conversation.

It is noticed that any inadvertent noise during this stage is apt to evoke in those who have been subjected to air raids responses of terror such as would follow explosion of a bomb.

Forcible restraint is liable to initiate or aggravate a patient's struggles. This too is the result of stimuli evoking in the aggressive the response which might be expected. Until the patient has entered the third stage of anæsthesia a soothing word or a pat on the arm is often more effective than vigorous handling. If however a struggling patient

considerably, e.g. for tooth extraction muscular relaxation is not essential (the mouth being kept open by a prop), and it can be performed at the very top of plane 1—in fact, as is well known, it occasionally takes place inadvertently at the bottom of stage II without deleterious effect on the patient. Examples of other operations which can be performed in plane 1 are amputations, operations on the chest and on the brain, and any other procedures not requiring marked muscular relaxation. Operations in the abdominal cavity however, necessitate profound anæsthesia to abolish the protective reflex spasm of the abdominal muscles. The reflexes from the upper abdomen are more vigorous than from the lower, and it can be stated as a generalisation, that plane 2 is necessary for lower abdominal surgery (e.g. appendicectomy) and plane 3 for most operations in the upper abdomen (e.g. excision of the gall-bladder).

It is evident that the assessment of the depth of anæsthesia, judged by the signs just described is made by reference to the kind and degree of muscular response present at different stages. In other words the signs of general anæsthesia are muscular signs. Correct assessment of the depth of general anæsthesia is impossible if a muscular relaxant e.g. *d* tubocurarine chloride is used in conjunction with the general anæsthetic. It is important for the anæsthetist using relaxant drugs to appreciate this and he must ensure that general anæsthesia is established before such drugs are given and must continue to administer sufficient general anæsthetic to maintain unconsciousness (p. 193). When such drugs are used general anæsthesia no deeper than first plane need be maintained even for abdominal operations, since the relaxation required is produced by other means.

#### REFERENCES

- (1) Guedel A. E. 1937 *Inhalation Anesthesia* New York.
- (2) Human J. U. 1938 *The Secrets of Blind Intubation and the Signs of Anæsthesia* London 21.



divided into four planes. Plane 1 is characterised by progressive decrease both in the range of excursion of movements of the eyeballs and in the rapidity of these movements until at the bottom of the plane the eye comes to rest in the central position where it remains throughout the deeper planes. The range of eyeball movement is represented on the chart by crosses. Plane 2 is dealt with at the end of this paragraph. At the upper boundary of plane 3 there begins a progressive decrease in thoracic respiration. Paralysis of the intercostal muscles increases until, at the bottom of the plane, respiration is carried on solely by the diaphragm. Plane 4 is marked by complete paralysis of the thoracic muscles and a progressive diminution in the activity of the diaphragm and at the bottom of this plane respiratory effort is absent because of paralysis of the respiratory centre from overdose of the anæsthetic drug. Plane 2 by the process of exclusion is characterised by central immobile eyeballs and complete functioning both of the intercostal muscles and of the diaphragm.

In the fourth stage (D-E) that of respiratory paralysis the heart still beats and the patient will remain alive if kept adequately oxygenated by artificial respiration. By continuing artificial respiration enough anæsthetic may be excreted to allow the respiratory centre to recover and initiate respiratory movements on its own account. E marks the level of cardiac arrest. A few cases are on record where through massage or needling of the cardiac muscle the heart beat has been restored and the patient revived even after this level has been reached.

Guedel has estimated that the minimum times in minutes taken by different inhalation anæsthetics to reduce a patient to respiratory arrest are roughly as follows: ether 10-30, chloroform 4-12, ethyl chloride 1-4, cyclopropane  $\frac{1}{2}$ -3. No matter what the drug or its method of administration the time depends on the rate at which it is given and on the rate of its absorption. Animal experiment shows that a drug such as thiopentone if rapidly injected can produce respiratory arrest in the circulation time of the blood.

On general principles the patient should be reduced to the minimal state of depression necessary for the operation in hand to be performed to the surgeon's satisfaction. The shorter and the less profound the anæsthesia the smaller is the risk to which the patient is exposed during operation and the less the likelihood of post-operative complications. In other words the lighter and shorter the anæsthesia the better.

The depth of anæsthesia necessary for different operations varies

verified in a patient who is to have a tooth extracted, by administering nitrous oxide with say 12 per cent oxygen, and then allowing the dentist to begin. The great majority of normal patients in these circumstances will show considerable response to the stimulus. Some will scarcely lose consciousness and will give a vivid and unflattering account of the experience.

Guedel<sup>1</sup> points out that nitrous oxide given with sufficient oxygen to satisfy basal metabolic needs can depress a patient in average health only a certain amount—from the line A to about the level (a), a man of 30 in normal health starts anæsthesia from the metabolic rate level (p. 89) corresponding to his age, and he will be depressed to (b), the same man stimulated by pain and fear will have his starting point raised above the normal metabolic rate line and can be depressed by a similar mixture of nitrous oxide and oxygen only to the level (c) which is just below the level at which consciousness is lost. If it is desired to anæsthetise the patient in this condition with nitrous oxide as the sole anæsthetic anæsthesia can be achieved only by reducing the oxygen in the mixture. In a robust patient this deprivation of oxygen is often the main factor in the resulting unconsciousness. The signs of anæsthesia are then complicated, and possibly overshadowed by the signs of acute asphyxia. The more the oxygen is reduced the more are the signs of anæsthesia masked by those of asphyxia. Because of the introduction of this asphyxial element, which has no place in other anæsthetics the utility of Guedel's chart for depth of anæsthesia is invalidated and the anæsthetist must rely on new landmarks.

It is essential to understand during the administration of nitrous oxide which signs indicate that the patient is (i) adequately anæsthetised and (ii) dangerously asphyxiated.

The beginner often finds it difficult to know both when a patient is unconscious and when he is ready for operation. To judge the moment precisely needs much experience. It is easy to subdue the patient until he is obviously deeply anæsthetised but by doing so refinements of anæsthesia are missed. It is impossible to give any fixed rules for determining the percentages of nitrous oxide and oxygen which will produce the correct depth of anæsthesia for any particular patient or for any particular operation.

The chief signs of nitrous oxide anæsthesia in order of importance are respiratory signs muscular signs eye signs and colour changes.

## CHAPTER VII

### SIGNS OF NITROUS-OXIDE ANÆSTHESIA

ALMOST every anæsthetic can reduce a patient to the point of death even though he is abundantly supplied with oxygen. The one notable exception is nitrous oxide the lack of potency of which is not sufficiently appreciated. If this gas mixed with enough oxygen to meet basal metabolic requirements is administered to an average fit patient who is unpremedicated not only is it impossible to reach the point of death but it is often difficult to subdue him sufficiently to allow even a minor operation to be performed. This can easily be

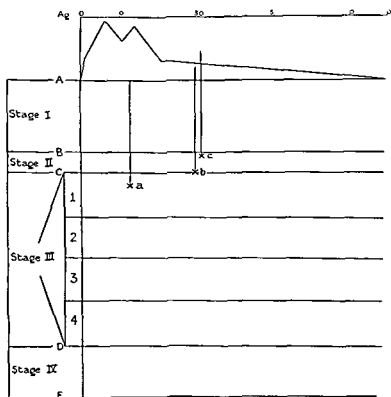


FIG 16

verified in a patient who is to have a tooth extracted by administering nitrous oxide with say, 12 per cent oxygen and then allowing the dentist to begin. The great majority of normal patients in these circumstances will show considerable response to the stimulus. Some will scarcely lose consciousness and will give a vivid and unflattering account of the experience.

Guedel<sup>1</sup> points out that nitrous oxide given with sufficient oxygen to satisfy basal metabolic needs can depress a patient in average health only a certain amount—from the line A to about the level (a) a man of 30 in normal health starts anæsthesia from the metabolic rate level (p. 89) corresponding to his age and he will be depressed to (b) the same man stimulated by pain and fear will have his starting point raised above the normal metabolic rate line and can be depressed by a similar mixture of nitrous oxide and oxygen only to the level (c), which is just below the level at which consciousness is lost. If it is desired to anæsthetise the patient in this condition with nitrous oxide as the sole anæsthetic, anæsthesia can be achieved only by reducing the oxygen in the mixture. In a robust patient this deprivation of oxygen is often the main factor in the resulting unconsciousness. The signs of anæsthesia are then complicated, and possibly overshadowed by the signs of acute asphyxia. The more the oxygen is reduced the more are the signs of anæsthesia masked by those of asphyxia. Because of the introduction of this asphyxial element, which has no place in other anæsthetics the utility of Guedel's chart for depth of anæsthesia is invalidated and the anæsthetist must rely on new landmarks.

It is essential to understand during the administration of nitrous oxide which signs indicate that the patient is (i) adequately anæsthetised and (ii) dangerously asphyxiated.

The beginner often finds it difficult to know both when a patient is unconscious and when he is ready for operation. To judge the moment precisely needs much experience. It is easy to subdue the patient until he is obviously deeply anæsthetised but by doing so refinements of anæsthesia are missed. It is impossible to give any fixed rules for determining the percentages of nitrous oxide and oxygen which will produce the correct depth of anæsthesia for any particular patient or for any particular operation.

The chief signs of nitrous oxide anæsthesia in order of importance are respiratory signs, muscular signs, eye signs and colour changes.

## Respiratory Signs

The onset of automatic respiration is with nitrous oxide as with other anæsthetics the sign that surgical anæsthesia has been reached. In an average induction the breathing becomes automatic after about twelve full breaths of nitrous oxide have been taken—that is after about 40 seconds. There is however this difference between the onset of surgical anæsthesia with nitrous oxide and with less pleasant inhalation anæsthetics such as ether and ethyl chloride—with the former induction is not oppressive so that the patient who is not nervous breathes regularly from the start and since the early stages of anæsthesia are passed through rapidly only the highly nervous show any irregularity of the respiratory rhythm during induction. Indeed if during this period the patient controls himself well there will be little if any appreciable change in the character of respiration as unconsciousness supervenes. This is in marked contrast to the obvious transition from irregular to automatic respiration which occurs at the onset of surgical anæsthesia when a less pleasant induction is produced as by ether or ethyl chloride. In dental anæsthesia the presence or absence of nasal breathing is an all important indication of the depth of anæsthesia. When nitrous oxide is given nasally a one way valve on the nose piece through which expirations are audible allows any change in the character of the respiration to be detected at once. The first thing to be learnt is that unless respiration is regular and is entirely nasal the patient is not sufficiently anæsthetised to tolerate any operative interference. These must be regarded as negative signs since in a co operative patient respiration may show these features from the beginning of induction. When such a patient is in the analgesic stage the respirations may lead the anæsthetist to think that anæsthesia has been reached and he may allow the dentist to begin work. If the patient is very placid he may continue to breathe through his nose even though he is aware of the extraction. It is obviously undesirable for extractions to be performed at this stage but this is not a grave mistake if the patient is sufficiently placid because as a rule when he recovers he merely remarks as a matter of interest that he knew of the removal of the tooth. The presence of nasal breathing does not prove that the patient is adequately anæsthetised *but its absence shows that he is not*.

Surgical anæsthesia sufficient for minor operations such as incision of an abscess or dental extractions is characterised by automatic respirations resembling those of natural sleep though somewhat deeper and quicker. At a slightly deeper level of anæsthesia there may be a gentle snore or slight catch in inspiration and the presence of either

of these more positive signs is a confirmation that anaesthesia is deep enough for minor surgery

In a normal or plethoric patient deepening anaesthesia is marked by an increase in the frequency of respiration followed by a change in its character. The free, natural respirations give place to snatchy, irregular, guttural, or sobbing breaths. These abnormal respirations are due to oxygen lack which causes irregular, jerky, unco-ordinated action of the laryngeal muscles, and are seen only in the robust who by this time, show other confirmatory signs of unconsciousness. At a deeper stage still there is a tendency for inspirations to be short and sobbing whilst expirations become markedly prolonged

### Muscular Signs

Since muscular relaxation results from deep anaesthesia it is seldom a feature of nitrous-oxide anaesthesia. Fortunately relaxation is not essential for a minor operation such as the opening of an abscess nor for dental extractions because here the mouth is propped open beforehand. During induction the patient may make movements which are purposeful. For example he may cross his legs or fold his arms to make himself more comfortable; he may attempt to push away the operator to show that he is not yet anaesthetised; or he may put up his hand to adjust the mask or nose piece because it is uncomfortable. During light anaesthesia a painful stimulus may lead to movement or a moan which shows that anaesthesia is inadequate. The movement such as an attempt to grasp the operator's hand is usually purposeful and the moan is one of pain. This movement and moan are generally easily distinguishable from those of deep anoxia in which condition the movements are purposeless and unrelated to stimuli and the moan is a phonation which does not give the impression of being caused by pain. If the oxygen intake is markedly reduced the tone normally present in muscle is readily superseded by spasmodic contraction. This spasm occurs first in the small muscles of the tongue, but is here rarely noticed and its early manifestation can be more conveniently observed in the eye where spasm of the intrinsic muscles pulls the eye out of centre. If the eyeball be examined at this stage, it will usually be found moving from side to side. At a later stage it becomes fixed in an eccentric position generally downwards. Twitching and spasmodic contractions of the larger muscles of the arms and legs follow. Later there may be opisthotonos and inspiratory crowing caused by spasm of the adductor muscles of the larynx. If deprivation of oxygen is continued still further this spasm gives way to complete muscular relaxation quickly followed by death.

## Eye Signs

Eye signs are helpful to a beginner but the experienced anæsthetist comes to use them less and less until finally he rarely, if ever inspects the eye

Early in induction if the eyelid is drawn back it will be found resistant (fig 17) The anæsthetist should never inspect the patient's



FIG 17

eye at this stage If unconsciousness has not been reached the lifting of the eyelid may be construed as a prelude to premature operation and so alarm the patient A patient so disturbed may begin to breathe through his mouth or may move his arms to indicate that he is not unconscious If the anæsthetist wishes to inspect the eyeball he should make reasonably sure before he

lifts the lid that the patient is sufficiently anæsthetised to be quite unconscious

If the eye appears to be able to focus (fig 18) unconsciousness has not been reached The eye soon loses this look of intelligence and it is often possible to tell solely by watching the eye the time at which consciousness is lost Later the eyeball develops a quick horizontal movement the rapidity and excursion of which diminish with deepening anæsthesia

The characteristic eye movements of nitrous oxide anæsthesia are all manifestations of muscular spasm due to oxygen lack



FIG 18

In a healthy patient anæsthesia deep enough for minor operations is attained before the eyes become fixed out of centre but if the administrator wishes to make doubly sure that the patient is adequately anæsthe

tised, he should continue until this eccentric fixation is reached (fig 19). The degree of anoxæmia at this stage is not enough to cause marked spasm of the larger muscles of the body, and if anæsthesia is maintained at this level any small operation not requiring muscular relaxation (e.g. dental extraction) may now be performed. If the muscles of the eye are all in spasm, the action of the strongest will predominate. That the eye is generally pulled downwards has been attributed perhaps unconvincingly, to the fact that the inferior rectus muscle is more strongly developed than the others because it is the muscle in most frequent use, pulling the eye downwards as in reading.

In deep nitrous-oxide anæsthesia not only are the eyes fixed eccentrically but in addition the pupils are widely dilated. The size of the pupil is useless as a



FIG 19

guide because it is often dilated throughout in the early stages from fear and excitement, in the later from anoxia. Extreme dilatation of the pupil does indeed denote serious anoxia but by the time it appears other warning signs are present.

### Colour Changes

Cyanosis as a sign of anæsthesia is unreliable (Chapter V). Its presence is no indication of danger nor is its absence an assurance of safety. In the average patient however when unpremedicated or only mildly premedicated as is usual in the outpatient operating theatre or the dental surgery the onset of cyanosis roughly coincides with the attainment of the correct depth of anæsthesia for operation. This has led perhaps instinctively to the use of colour change as a rough guide even by the experienced. The anæsthetist, while relying primarily on the automatic character of the respirations nevertheless accepts the colour change as a confirmatory sign of unconsciousness. It is however the last sign on which reliance should be placed and a reminder may be given here that in severe anæmia it cannot even occur.



## Eye Signs

Eye signs are helpful to a beginner, but the experienced anæsthetist comes to use them less and less until finally he rarely if ever inspects the eye

Early in induction if the eyelid is drawn back it will be found resistant (fig 17) The anæsthetist should never inspect the patient's



FIG 17

eye at this stage If unconsciousness has not been reached, the lifting of the eye lid may be construed as a prelude to premature operation and so alarm the patient A patient so disturbed may begin to breathe through his mouth or may move his arms to indicate that he is not unconscious If the anæsthetist wishes to inspect the eyeball he should make reasonably sure before he

lifts the lid that the patient is sufficiently anæsthetised to be quite unconscious

If the eye appears to be able to focus (fig 18) unconsciousness has not been reached The eye soon loses this look of intelligence and it is often possible to tell solely by watching the eye the time at which consciousness is lost Later the eyeball develops a quick horizontal movement the rapidity and excursion of which diminish with deepening anaesthesia



FIG 18

The characteristic eye movements of nitrous oxide anaesthesia are all manifestations of muscular spasm due to oxygen lack

In a healthy patient anaesthesia deep enough for minor operations is attained before the eyes become fixed out of centre but if the administrator wishes to make doubly sure that the patient is adequately anæsthe-

tised, he should continue until this eccentric fixation is reached (fig 19). The degree of anoxæmia at this stage is not enough to cause marked spasm of the larger muscles of the body, and if anæsthesia is maintained at this level any small operation not requiring muscular relaxation (e.g. dental extraction) may now be performed. If the muscles of the eye are all in spasm, the action of the strongest will predominate. That the eye is generally pulled downwards has been attributed perhaps unconvincingly, to the fact that the inferior rectus muscle is more strongly developed than the others because it is the muscle in most frequent use pulling the eye downwards as in reading.

In deep nitrous-oxide anæsthesia not only are the eyes fixed eccentrically but in addition the pupils are widely dilated. The size of the pupil is useless as a



FIG 19

guide because it is often dilated throughout in the early stages from fear and excitement in the later from anoxia. Extreme dilatation of the pupil does indeed denote serious anoxia but by the time it appears other warning signs are present.

### Colour Changes

Cyanosis, as a sign of anæsthesia is unreliable (Chapter V). Its presence is no indication of danger nor is its absence an assurance of safety. In the average patient however when unpremedicated or only mildly premedicated as is usual in the outpatient operating theatre or the dental surgery, the onset of cyanosis roughly coincides with the attainment of the correct depth of anæsthesia for operation. This has led perhaps instinctively to the use of colour change as a rough guide even by the experienced. The anæsthetist while relying primarily on the automatic character of the respirations nevertheless accepts the colour change as a confirmatory sign of unconsciousness. It is however the last sign on which reliance should be placed, and a reminder may be given here that in severe anæmia it cannot even occur.

## THE ANÆMIC PATIENT

Analysis of the signs of deep nitrous-oxide anæsthesia in the normal patient e.g. the snatchy jerky respirations, the twitching muscles the eccentric eye reveals that they are almost entirely due to muscular spasm resulting from oxygen lack. It cannot be emphasised too strongly that signs which depend on anoxial spasm of muscles may be entirely absent in anæmic subjects as also in patients heavily pre-medicated those convalescent from severe debilitating illnesses such as influenza and in those with exophthalmic goitre. The respirations of the anæmic patient are equally atypical. As anæsthesia deepens they become shallower and shallower. Respiratory effort diminishes progressively and the patient simply 'fades away' without becoming cyanosed or jactitating. Depression of respiratory activity and absence of muscular spasm are striking features of nitrous-oxide anæsthesia in this type of patient. If the anæsthetist waits for the onset of irregular jerky respiration, for the eyeballs to be pulled eccentrically for jactitation or for cyanosis as signs of anæsthesia he will soon be confronted with a patient *in extremis*. At this point, the pupils of the centrally fixed eyeball are widely dilated and an ashen grey colour further contributes to the patient's alarming appearance. Unless artificial respiration is promptly applied death quickly ensues.

Soon after the commencement of induction with nitrous oxide respiration becomes automatic. Provided sufficient oxygen is now given satisfactory anæsthesia can be maintained without depression of respiration or asphyxial dilatation of the pupil.

### Pulse

The pulse is of little value as a guide to  $N_2O$  anæsthesia. Early it may be quickened from excitement or fear while in asphyxia it becomes slow full and bounding. The significance of both may be erroneously interpreted, the former as heart disease the latter as a reassuring sign. By the time the asphyxial pulse is noticeable other conspicuous signs indicate that oxygen lack is excessive. The onset of any cardiac irregularity at this stage is an urgent indication for the administration of oxygen.

### REFERENCE

- (1) Guedel A. E. 1937 *Inhalation Anesthesia* New York 68

## CHAPTER VIII

INDICATIONS FOR LOCAL OR GENERAL  
ANÆSTHESIA IN DENTISTRY

In deciding upon the type of anæsthesia to employ for a dental operation it is well to remember that general anæsthesia is now so safe that it can reasonably be assumed that a patient who is fit for a local anæsthetic is also fit for a general. The solitary exception to this generalisation is the patient suffering from œdema of the glottis (pp 301-304)

The responsibility for deciding between local and general anæsthesia is often borne by the dentist. Conditions in private practice are such that this choice is not the clear-cut problem which it is at hospital where the question of the anæsthetist's skill is not a deciding factor. In cases where the choice lies between local anæsthesia and a general anæsthetic given by a family doctor who is far from skilled in this speciality local anæsthesia may well be selected when ideally general anæsthesia would be preferable. Thus, subject to there being no preponderant contra indication to either local or general anæsthesia the dentist will find that the technical skill of the anæsthetist available inevitably plays a large part in determining his choice.

Assuming that the choice lies between local anæsthesia and *skilled* general anæsthesia the following considerations will help the dentist in making his decision.

Considerable weight should be attached to any decided preference for either local or general anæsthesia which the patient may express since any physical disability to which he is subject is often of far less importance than his mental attitude towards anæsthesia. Far too frequently a patient is persuaded against his will to submit to a type of anæsthesia which he dreads. One patient may be apprehensive of general anæsthesia because he dreads the idea of losing consciousness while another may wish for general anæsthesia so that he will know nothing about it. Usually men prefer local women general anæsthesia.

No more definite reason than personal inclination often determines a dentist to choose one particular form of anæsthetic. A dentist may prefer to extract on a patient sitting upright and on this account recommend local anæsthesia for a major dental operation so that he

## THE ANÆMIC PATIENT

Analysis of the signs of deep nitrous oxide anæsthesia in the normal patient e.g. the snatchy jerky respirations, the twitching muscles the eccentric eye reveals that they are almost entirely due to muscular spasm resulting from oxygen-lack. It cannot be emphasised too strongly that signs which depend on anoxial spasm of muscles may be entirely absent in anæmic subjects as also in patients heavily pre-medicated those convalescent from severe debilitating illnesses, such as influenza and in those with exophthalmic goitre. The respirations of the anæmic patient are equally atypical. As anæsthesia deepens they become shallower and shallower. Respiratory effort diminishes progressively and the patient simply fades away, without becoming cyanosed, or jactitating. Depression of respiratory activity and absence of muscular spasm are striking features of nitrous-oxide anæsthesia in this type of patient. If the anæsthetist waits for the onset of irregular, jerky respiration for the eyeballs to be pulled eccentrically for jactitation or for cyanosis as signs of anæsthesia he will soon be confronted with a patient *in extremis*. At this point the pupils of the centrally fixed eyeball are widely dilated and an ashen grey colour further contributes to the patient's alarming appearance. Unless artificial respiration is promptly applied death quickly ensues.

Soon after the commencement of induction with nitrous oxide respiration becomes automatic. Provided sufficient oxygen is now given satisfactory anæsthesia can be maintained without depression of respiration or asphyxial dilatation of the pupil.

### Pulse

The pulse is of little value as a guide to  $N_2O$  anæsthesia. Early, it may be quickened from excitement or fear while in asphyxia it becomes slow full and bounding. The significance of both may be erroneously interpreted the former as heart disease the latter as a reassuring sign. By the time the asphyxial pulse is noticeable other conspicuous signs indicate that oxygen lack is excessive. The onset of any cardiac irregularity at this stage is an urgent indication for the administration of oxygen.

### REFERENCE

- (1) Guedel A. E. 1937 *Inhalation Anesthesia* New York 68

heal more quickly after local anæsthesia than after inefficient general anæsthesia because during the former there is more time for extractions to be performed deliberately and with less laceration of the mucous membrane and less damage to the alveolus

9 Removal of infected teeth for systemic disease —There is a clinical impression that local anæsthesia is followed by a graver exacerbation of the patient's symptoms than if the teeth are extracted under general anæsthesia

10 If the dentist considers that the patient's temperament makes general anæsthesia advisable —It is unwise to recommend local anæsthesia for the super-critical patient who is only too keen to find grounds for complaint. If under local anæsthesia the tooth should break the patient cannot fail to appreciate that it has to be removed in fragments. If the extraction takes a long time to complete, the patient may consider this to be evidence of the dentist's inefficiency. For such a patient general anæsthesia is preferable

11 If the dentist particularly wishes to work with a general anæsthetic

## INDICATIONS FOR LOCAL ANÆSTHESIA

1 If there is any doubt about the anæsthetist's skill —It has been pointed out that the dentist who cannot rely on skilled general anæsthesia may in self defence choose local. The patient seldom attributes an incomplete extraction to inadequate anæsthesia. If the dentist cannot rely on good general anæsthesia he is justified in advising local particularly where extractions have to be made with deliberation—e.g. when the teeth are exostosed or where the apex of a tooth has to be preserved uncontaminated for bacteriological culture

2 If the patient particularly requests local anæsthesia

3 Extractions during certain acute illnesses—e.g. bronchitis

4 Where extractions have to be made on a patient who has recently had a meal

5 Occasionally in patients with phthisis —This disease is not a contra indication to administration of  $N_2O$  for simple extractions, but the risk of initiating a violent bout of coughing by the passage of an endotracheal tube often makes it preferable to use a local anæsthetic for such major dental operations as would normally be done under general anæsthesia. When a phthisical patient has particularly requested general anæsthesia for dental operations we have produced unconsciousness first with avertin and then after abolishing the cough

can work in the position in which he feels most at ease. This preference should not be allowed to weigh too heavily since a dentist can soon accustom himself to operating on recumbent patients. In any case it is easy and safe to administer a general anæsthetic even for a prolonged operation on a patient who is sitting up. For this purpose an armchair may be used or a dental chair can be taken to a private house or nursing home. Similarly for a prolonged difficult extraction some dentists prefer local anæsthesia because they like to have the active co operation of the patient but in these days such co operation can well be dispensed with by using a modern head rest and tongue and other tissue retractors.

## INDICATIONS FOR GENERAL ANÆSTHESIA IN DENTISTRY

1 **The presence in the mouth of a general infection or of a local infective process**—A local anæsthetic must not be used here because of the danger of its carrying infection from the diseased region into healthy gum or bone. Apart from this imperative contra indication it must be remembered that any local anæsthetic interferes to a greater or lesser extent with the circulation in the region affected and by thus lowering tissue resistance favours bacterial invasion.

2 **Multiple extractions** from the various quadrants of the mouth.

3 **Operations on young children**, who on the whole are unco operative.

4 **Fractures of the jaw**.

5 **If the patient particularly requests general anæsthesia**.

6 **Where considerable force must be applied**—Under local anæsthesia a nervous patient often finds it difficult to discriminate between force and pain. If it is anticipated that a considerable amount of force will have to be exerted a nervous patient should be advised to have his tooth extracted under general anæsthesia.

7 **For long operations**—General anæsthesia is usually preferred for such operations as the removal of an impacted wisdom tooth, alveolectomy and excision of a cyst for if a local anæsthetic is used not only is the patient likely to become fatigued but the effect of the anæsthetic is liable to wear off before the operation has been completed.

8 **Cases where post-operative pain is specially to be avoided** and quick healing is particularly desirable. It is generally considered that ischæmia following local anæsthesia predisposes to after-pain and to slow healing of the sockets. In fact wounds often

## CHAPTER IX

### CHOICE OF GENERAL ANÆSTHETIC

AFTER the decision has been made to employ general anæsthesia the means by which this is to be attained must be selected. The extent and nature of the operation the anæsthetic agent and technique available and the patient's temperamental and physical make up must all be considered.

Safety is of greatest importance and is always in the mind of the patient the anæsthetist and the surgeon. Too much attention is often paid to the supposed safety or danger of a particular narcotic drug as indicated by the available statistics. *An anæsthetic is made safe or dangerous by the one who administers it.* If the anæsthetist is ready to deal with any untoward occurrences is not venturesome beyond his experience and does not trust to luck the risks to which his patient is exposed will be negligible. Avoidance of anæsthetic accidents undoubtedly depends far more on the experience care and skill of the anæsthetist than on the choice of any particular anæsthetic agent.

Statistics can be entirely misleading as a guide to the relative safety of the various anæsthetics. Figures may be produced to show that one death has occurred in say 50 000 administrations of nitrous oxide 20 000 of ether and 10 000 of ethyl chloride. Such figures are valueless because they do not take into consideration such important factors as the skill and experience of the anæsthetist the state of health of the patient the nature of the operation its duration and severity and they do not show how many times an operation had to be abandoned on account of difficulties in connection with the maintenance of anæsthesia. This last factor is significant particularly in dental work where every dentist is familiar with the case in which it has been impossible to complete extractions because the length of anæsthesia or the facilities afforded by nitrous oxide were inadequate. Such a case would be classified for statistical purposes as a successful anæsthetic (i.e. not fatal) although it failed completely to enable the operation for which it was given to be performed.

Taking surgery as a whole it is found that the mortality rate of an operation is closely connected with its severity. To argue from statistics that nitrous oxide is a safer anæsthetic than ether is unjustified.



reflex by cocaineising the larynx and trachea we have passed an endotracheal tube and continued the anæsthesia with  $N_2O$

**6** As an excuse, when the dentist is requested to perform a difficult operation under general anæsthesia in the patient's home, where conditions for surgery are far from perfect Every experienced practitioner is familiar with the patient who has a rooted prejudice against institutional treatment and argues that the expense is unnecessary and that the short period of convalescence could be spent at home—a plea frequently advanced by the woman who considers herself indispensable to the household These excuses are made the more strongly because of a mistaken idea that any operative procedure on the teeth no matter how extensive is simple The patient refuses to enter a nursing home and asks for the operation to be done at home The suggested operating table is often the kitchen table or even a sagging bed Operating on the patient in an armchair or in a dental chair taken to the house solves some of the problems but handicaps still remain The lighting is nearly always poor but the most serious disadvantage of all is that it is almost impossible to ensure asepsis After treatment in the patient's home is moreover frequently much handicapped In such cases the dentist is justified in urging local anæsthesia in his own surgery where asepsis will be perfect the operator's own instruments are at hand and the lighting is good Even after formidable dental operations under local anæsthesia the patients are often fit to visit the dentist's surgery for after treatment This indication for local anæsthesia does not exist where an enlightened patient agrees to enter a hospital or a good nursing home for his operation

**7** If the dentist himself has a special preference for working with local anæsthesia—Some dentists prefer local anæsthesia because the adrenaline added to the injected solution assists in checking hæmorrhage Local anæsthesia need not be chosen on this score alone since for prolonged operations a local anæsthetic solution containing adrenaline can be injected immediately after general anæsthesia has been induced

**8** Financial considerations—With an indigent patient on whom a prolonged operation is contemplated it is desirable to take into consideration also the fact that local anæsthesia is more economical since it saves the anæsthetist's fee

anæsthetic for the period of the operation itself, many consider that its slightly irritant effects on the lungs are enough to aggravate the existing chest condition. They argue that the possibly greater risk of cyclopropane anæsthesia at the time of operation is justified by the diminished chances of post operative complications and in fact, we ourselves use cyclopropane under these conditions. It is, however, our opinion that the dangers of ether in such cases have been much exaggerated and we believe that provided the patient has been properly premedicated, and that an uninterruptedly free airway is maintained, then ether is no more likely to initiate a pulmonary complication or to aggravate an existing chest condition than is any other anæsthetic.

It is still surprisingly common for the surgeon to ask a physician for his opinion on what would be a suitable choice of anæsthetic for a poor risk patient. It is extraordinary that a surgeon who realises how much the safety of his patients depends on the experience and skill of the anæsthetist should feel that the opinion of a physician who may never enter an operating theatre will be helpful in selecting an anæsthetic. This is not to say that consultation on any of these matters is not advisable providing the anæsthetist is present with the physician and surgeon when the question of choice of anæsthetic is to be considered but all too frequently the surgeon calls in the physician only. The physician by consulting with anæsthetists on doubtful cases will gradually learn something of the problems of anæsthesia and, when he does this his opinion as to a suitable anæsthetic will be of value. It is at present extremely rare for a physician's opinion in such matters to be at all helpful. Anæsthetists recognise the truth of this statement but for economic reasons this view is rarely publicly expressed. If the surgeon is not satisfied to leave the whole question of the anæsthetic to the anæsthetist and if he feels that he wants a second opinion he should call in a second *anæsthetist* so that he may have the benefit of the advice of someone who appreciates the problems involved. The best way to acquire an understanding of patients' reactions physical and mental to anæsthesia is to administer anæsthetics as often as possible and it is significant that when an anæsthetist is faced with a difficult problem in anæsthesia he rarely if ever even considers consulting a physician but obtains the advice of anæsthetists of experience who have dealt with similar cases in practice.

It is also not at all unusual for patients who are anxious about themselves or those who are physically unfit and who have been under the care of a consulting physician or of the family doctor for some time to enquire of their advisers whether general anæsthesia

Such figures are obtained only because nitrous oxide is rarely chosen for severe operations. It is in fact used almost exclusively for short and trivial operations and then often on robust patients in whom a fatal issue is extremely improbable. Ether on the other hand is more frequently chosen for major operations the outcome of which may cause the anæsthetist grave anxiety. The difference between the safety of a weak anæsthetic such as nitrous oxide, and of a more potent one such as cyclopropane or ether must therefore be considered in relation to the conditions it is asked to provide. It is wrong to state categorically that nitrous oxide will be a safer anæsthetic than ether without knowing what operation is to be performed.

For minor operations such as tooth extraction nitrous oxide although much more convenient is in fact no safer than ether although the incidence of post-operative complications particularly vomiting is greater after the latter. For major operations ether is safer than nitrous oxide. Very many major operations cannot be performed under such a weak anæsthetic as nitrous oxide because satisfactory operating conditions cannot be produced unless this anæsthetic is pushed and the patient deprived of oxygen to such an extent that in the hands of the average anæsthetist the number of fatalities would be greater than if a more potent anæsthetic such as ether were used.

In abdominal surgery for example the provision of muscular relaxation is necessary for without it the surgeon is greatly handicapped at his work. An anæsthetic can be considered safe only if it produces muscular relaxation before the cardiac output and minute volume respiration are appreciably depressed. Since ether possesses this attribute it is justifiably popular in abdominal surgery. Cyclopropane is not so reliable. Only a slight increase in concentration of this drug above that necessary to produce muscular relaxation may be followed by respiratory arrest or cardiac irregularities. Similarly thiopentone *can* be used for abdominal surgery but a small increase in the amount of drug necessary to produce relaxation may cause profound respiratory depression. These considerations apply to general anæsthesia used alone. The coincident use of relaxant drugs enables muscular relaxation to be provided with only light levels of general anæsthesia (p. 193). Spinal anæsthesia provides extreme muscular relaxation but may not be desirable either because of the fall in blood-pressure which it causes or because it is unsuited to the patient's temperament.

The patient's general health must be taken into consideration. If he suffers from phthisis or bronchitis although ether may be the safest

*Case 4*—For this hospital patient suffering from rheumatic carditis the physician whose advice was asked as to what anæsthetic should be given wrote 'Fit for gas' on the case-sheet. There was no reason why the patient should not have a more potent anæsthetic and in fact nitrous oxide would have been unsuitable since anoxia in such a case should be avoided so that the anæsthetist used his own discretion and gave ether.

A point which must not be forgotten is that the mental attitude towards anæsthesia of a patient who carries a certificate forbidding general anæsthesia is not improved by the constant awareness of all that the certificate implies. Circumstances may arise in which anæsthesia is imperative and the psychological problems involved in anæsthesia are difficult enough without the addition of such unnecessary mental burdens.

The physician acts, as he thinks for his patient's good but the unwitting arrogance of his dogmatism betrays his ignorance of important fundamentals of anæsthesia. The result is that an anæsthetist has frequently been virtually forced to agree to the patient being deprived of the advantages of basal anæsthesia solely to comply with the unwarranted advice of a consulting physician.

Often the physician's choice of anæsthetic is one with which the anæsthetist does not agree and then the latter must either deny his patient the advantages of what his experience leads him to know is the correct anæsthetic or he must be prepared to face the economic repercussions which may result if he incurs displeasure through not following the advice of the physician whose opinion as an alleged expert has been sought. In practice the anæsthetist often does not spend time in disputing the choice advised but just as often deviates from it in what he actually administers. For instance advice as unsubstantial as the 'fit for gas' mentioned in *Case 4* is all too often written dogmatically upon the case sheet of an ill patient. The physician giving such an opinion has usually only a scanty knowledge of and experience in anæsthesia and has no appreciation of the limitations of nitrous oxide. He believes from what he has heard that nitrous oxide is the safest anæsthetic but we have already shown on what slender evidence this opinion is based. Indeed nitrous oxide may be advised when in fact it is actually contra-indicated sometimes because of the type of patient sometimes because of the nature of the operation to be performed. It is not surprising that the anæsthetist should not then follow the physician's advice but that he should quietly supplement the gas with cyclopropane or ether so that anæsthesia is

at some future date would be likely to be detrimental. A physician should be extremely guarded in his remarks on this subject. If he should give the impression that general anæsthesia is likely to be fatal or if he should suggest that certain anæsthetics would be dangerous his patient may consider that this is a proud distinction for it is undoubtedly true that some patients derive a certain macabre pleasure from a feeling that they are thus distinguished from their fellows. More usually however the patient is terrified of ever having to submit himself to general anæsthesia. The following four cases in our own experience are instances of the unhelpfulness of the advice of the physician on anæsthesia, the first three cases occurred in private the fourth in hospital practice.

*Case 1*—This woman aged 60 produced a certificate signed by a cardiologist stating that she must in no circumstances be given a general anæsthetic. This was the only information the certificate gave. It had been signed twelve years previously and had been a source of considerable anxiety to her through all these years. She suffered from an ovarian cyst which had so increased in size that operation had become imperative. Despite the fact that the greatly distended abdomen hindered her respiration when she was lying down she took a general anæsthetic well and made an uninterrupted recovery.

*Case 2*—A young woman presented a certificate from a physician to say that on account of 'heart disease' she was not to be given in any circumstances a general anæsthetic. On auscultation a presystolic murmur was audible but since she carried out a usual day's routine quite normally the anæsthetist had no compunction in giving her gas and oxygen for extraction of a tooth involved in an alveolar abscess for which it was considered undesirable to give a local anæsthetic. She also caused no anxiety whatever.

*Case 3*—Soon after the introduction of evipan we were requested by a distinguished physician not to give this drug to a patient whom we were to anæsthetise on the following day. When pressed for any reason why evipan should not be given his only answer was that he had heard that there had been a death in Peckham the week before from this drug! Our previous experience with earlier barbiturates (pernocton and nembutal) added to our experience with evipan in about a hundred cases persuaded us that it was particularly suitable for this nervous resistant young man as a means of induction before continuing with endotracheal anæsthesia for removal of an impacted wisdom tooth. Evipan was used for induction and the subsequent anæsthesia was uneventful.

One of the causes of post-operative depression is moving a patient from the dental surgery soon after consciousness is regained. A long operation of the type indicated, if carried out in hospital or a nursing home is attended with much happier after effects and recollections. In these circumstances ideal induction by basal anæsthesia can be obtained, and after the operation is over, the patient need not be roused. The patient's gratitude to the anæsthetist may be earned more easily under such conditions than in the dental surgery though the amount of skill exercised may be far less.

Each of the various agents used in producing general anæsthesia in the dental surgery has its own merits and limitations, and these will now be considered briefly. In a nursing home or hospital, appropriate basal anæsthesia can be used to modify the patient's reaction to anæsthesia. In the dental surgery patients can be premedicated only very lightly and the anæsthetist's main problem is that he is expected to provide a pleasant induction, tranquil anæsthesia and a quick recovery with freedom from after-effects for all his patients, despite their individual reactions to anæsthesia.

**Nitrous oxide**—This is the only anæsthetic which has a reasonable chance of satisfying all the above requirements. It is emphasised throughout this book that the anæsthetic properties of  $N_2O$  are feeble, but it owes many of its desirable qualities to this very fact and it is justifiably the most popular anæsthetic at present available for extractions from the average patient. In fact the number of administrations of this gas is far greater than of those of all other anæsthetics used in dentistry put together. Unconsciousness can be easily and pleasantly produced with  $N_2O$  recovery is prompt and the after effects are usually minimal—factors gratifying to both the patient and the busy dentist—but it is in the production and maintenance of adequate anæsthesia that the limitations of  $N_2O$  are appreciated. It is not on its own able to supply tranquil anæsthesia in the highly resistant patient. An anæsthetic emergency which was due solely to the incorrect choice of  $N_2O$  for an anæsthetic-resistant patient for dental extractions is described on p. 311.

Nitrous oxide is the only non explosive inhalation anæsthetic in common use. A dental surgery so often contains a naked flame that the use of several other anæsthetic gases, which but for their explosiveness would be of great value is precluded. For example though the potency of ethylene is not extreme it is enough to produce tranquil anæsthesia adequate for minor surgery and dental work even when mixed with 15-20 per cent of oxygen. Ethylene is however highly explosive and many deaths from explosions have been recorded.

attained mainly by the more potent agent. A happy outcome is regarded by everyone—except the anæsthetist—as justification of the physician's choice.<sup>1</sup>

If a physician objects to a particular anæsthetic he should be prepared to take full responsibility for anything untoward which may result from the course taken in deference to his objection. If he is not willing to take this responsibility he should leave the choice to the anæsthetist.

### Choice of General Anæsthetic in the Dental Surgery

Before considering the above question it is well to emphasise that the dental surgery is not the place of choice for administration of general anæsthetics for long operations. Quick recovery and lack of after effects rarely fail to satisfy the patient and these benefits follow anæsthesia of short duration. A long operation in the dental surgery even if the operative result is highly successful inevitably causes some degree of collapse and often a disappointed and dissatisfied patient. This is particularly so if the patient has previously had a quick recovery to normal following an easy extraction. He naturally assumes that the dentist and anæsthetist on the second occasion are unskilful and in the interests of all work which is expected to take a long time should not be embarked upon in the dental surgery.

A good general rule is not to anæsthetise in the dental surgery any case which the dentist estimates will take longer than ten minutes operating time. Sometimes however an extraction which was expected to take only a few minutes lasts much longer. The situation here is quite different and in such cases it may be kindness or good policy if the anæsthetist is sufficiently skilful to continue the nasal administration of nitrous oxide until the extraction is completed. There are alternatives neither completely satisfactory. The first is to abandon the operation in which case the patient must again submit to the procedure. the second is to continue the operation knowing that the patient will probably suffer unpleasant after effects which he would not anticipate. Which course should be adopted is dictated to some extent by the type of patient. If he is likely to blame the dentist for an incomplete extraction under the impression that it indicates inefficiency it might be entirely justifiable to proceed with the operation in the expectation of its successful completion. In such a case however the anæsthetist must not expect from the patient the appreciation to which his skilled assistance has entitled him. the patient will be acutely aware of his post-operative depression and will probably blame the anæsthetist for this part of his experience.

**Ethyl chloride**—This drug may be used alone or to supplement nitrous oxide when the latter proves inadequate as the sole anæsthetic. Although seldom necessary in the average patient over 8 or 9 years of age, it is of great value in such apparently divergent types as the very young child and the anæsthetic resistant adult. For the former ethyl chloride alone is commonly given (p 153), whilst for the alcoholic patient it is more commonly used to supplement nitrous oxide (pp 117-162). The chief disadvantage of ethyl chloride is the frequency of nausea and vomiting during the recovery period.

**Vinesthene (di-vinyl ether)**—The indications for the use of this drug are very similar to those for ethyl chloride. It has the advantage over the latter that in the period of recovery nausea, vomiting and collapse are rare.

Since these various drugs of known and proved value are available it is usually easy to choose appropriate anæsthesia for a particular case. The matter is not, however, uniformly simple and all the difficulties are not solved once the anæsthetic agent has been selected.

The experience of the administrator must be taken into consideration and while the general practitioner cannot be expected to master the many anæsthetic techniques used in general surgery, the choice in dentistry is more restricted and he can with practice become reasonably proficient in them all. The range of utility of any anæsthetic is extended by experience in its use. For example although with nitrous oxide the student will at first be successful only in straightforward cases familiarity will soon enable him to employ this anæsthetic successfully for cases presenting greater difficulties. Even a skilful anæsthetist is prepared to use supplementary anæsthesia for particularly difficult cases and the inexperienced will be wise to resort to this expedient rather than to try to force the maximum of usefulness out of nitrous oxide alone.



Although induction with nitrous oxide should be pleasant certain patients particularly those who have had a previous disagreeable anæsthetic experience dislike the induction of unconsciousness by any inhalation anæsthetic. For these, the advantages offered by the ultra short-acting barbiturates must be considered.

**Ether**—This is conceded to be a safe anæsthetic which affords excellent operating conditions. There are however, many objections to its use in the dental surgery. Induction of anæsthesia with it is a much longer and generally more unpleasant experience than is induction by nitrous oxide. Recovery is slower and is often followed by nausea and vomiting. The use of ether in small doses to supplement nitrous oxide for short operations in difficult patients and its use in general surgery are considered on pp 117 and 173.

**The ultra-short-acting barbiturates (e.g. thiopentone, hexobarbitone) for intravenous administration**—The main advantage of these drugs is that induction with them is pleasant and rapid. Their use is indicated in patients who fear anæsthesia or who are known previously to have been troublesome during induction and for anæsthetic resistant patients as a basal supplement to nitrous oxide anæsthesia. There are no contra indications to their use in small doses (e.g. 0.2–0.5 g of thiopentone) in the above types of case. There are however disadvantages in employing barbiturate anæsthesia alone for long dental operations. These are that in giving the larger doses which are then necessary technical difficulties arise in connection with the continuous intravenous administration of the drugs and although recovery from a small dose may be almost complete in a few minutes a fairly large dose may incapacitate the patient for hours.

For an operation lasting a few minutes both a pleasant induction to anæsthesia and a quick recovery can be obtained by producing unconsciousness with a minimal amount of thiopentone and then continuing with nitrous oxide administered nasally.

Although thiopentone is often ideal for the difficult (i.e. anæsthetic resistant) patient from whom extractions are expected to be easy this drug does not help at all in solving the problem of anæsthesia for a difficult extraction in an easy or normal patient. In fact thiopentone here should usually be avoided for if it is used as the sole anæsthetic for an extraction which lasts several minutes complete recovery from its effects may not take place for some hours. Some dentists erroneously regard thiopentone as a panacea for difficulties encountered in troublesome extractions and ask for it for this type of work in the normal patient whereas in fact its use is indicated for the *resistant* patient to be anæsthetised for easy extractions.

thetics than others. Different patients and even the same patient at different times show different degrees of alertness as also do normal healthy people. In other words, there are different levels of consciousness. Guedel points out that anything which increases the basal metabolic rate of the patient (expressed by his oxygen consumption) increases his reflex irritability and makes him more resistant to the effects of anæsthetics.

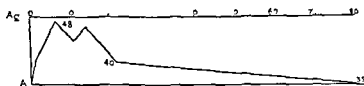


FIG 20 —The curve of the normal metabolic rate at different ages expressed in calories per hour per square metre of body surface. Line A of this diagram corresponds to line A in fig 16 p 68.

The metabolic rate varies with age. Thus in a patient aged 20 induction begins at a point more remote from surgical anæsthesia than in one aged 40 and consequently the amount of anæsthetic required to bring about surgical anæsthesia is greater in the younger patient. Apart from variations in the starting point of anæsthesia due to age anything which prevents the onset of normal sleep makes for increased resistance to the onset of anæsthesia. Thus pain, fear, apprehension, and excitement increase reflex irritability so that a given amount of anæsthetic fails to produce as much effect as it would in the absence of these factors.

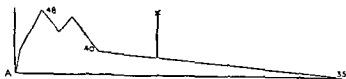


FIG 21

A patient of say 35 who is frightened of anæsthesia and who has been in pain for some time will start at X, a height much above level A, and will be correspondingly difficult to subdue.

\* Compiled by Will Shimer from the tables of Du Bois and Benedict published 1924 *Journ Amer Med Assn* 83 1736.

## CHAPTER X

### PRE-ANÆSTHETIC MEDICATION

BERNARD SHAW s gibe that anæsthesia spares us nothing but the knife<sup>1</sup> is fortunately not as true now as when it was written. Many modern improvements in surgical results are due to advances in the practice of anæsthesia. Nor is it only the relative immunity from grave issues which commands attention. The development of anæsthetic technique has not only diminished the hazard of operation but it has reduced to a minimum the terror and discomfort of the patient. Not many years ago the patient's preparation for an operation consisted mainly in the administration of a vigorous purge. After a disturbed night he walked in a more or less apprehensive state of mind to the operating theatre which even in happier circumstances, is a terrifying sight to most laymen. After he had climbed on to the operating table he had the unpleasant experience of having a mask applied to his face and then inhaling a strange and often distasteful vapour. It is not surprising that such physical and mental chastening was often followed in major surgery by post-operative shock which might well turn the scale against the patient and even after a minor operation often left a deep mental impression the gravity of which it was difficult to evaluate. In contrast to all this the patient is now a days assured of a good night's sleep before operation and he can be given a basal anæsthetic while still in bed before he is taken to the scene of the operation. From the drowsiness which supervenes he wakes up in his own bed to find with surprise and pleasure that the operation is a thing of the past and this early peacefulness of mind contributes materially to speedy and uninterrupted convalescence. Such modern improvements notwithstanding anæsthesia is still dreaded because it is commonly experienced in association with pain acute illness sleeplessness financial worries and enforced absence from work.

It is helpful to consider anæsthesia as the summation of the effects of the premedication and of the anæsthetic proper. The student who desires to master the technique of anæsthesia should not overlook the important part played by premedication in achieving good results.

Guedel<sup>2</sup> has rendered a great service to anæsthetists by explaining the well known fact that some patients are more resistant to anæ-

the student who has been rendered unconscious for a few minutes experimentally. The latter naturally chooses a time when he is feeling particularly well, the period of anæsthesia is short, he chooses his own anæsthetic and carefully selects an anæsthetist in whom he has complete confidence. He has none of the anxiety produced by the anticipation of an operation and, since no operation is performed, post-anæsthetic depression is reduced to a minimum.

It is rare to find anyone apprehensive of a first anæsthetic, the need for sedation is often increased by a previous acquaintance, generally made in childhood, with some unpleasant experience of anæsthesia. Since anæsthesia has improved so rapidly during the past few years, the fear of it will probably gradually be met with less and less frequency but it must not be forgotten that even under the best conditions an inhalation anæsthetic can rarely be described as pleasant. We may add that addiction to anæsthetics is practically unknown!

A patient's fears are largely created by an active imagination and prolonged psychological treatment might be necessary to enable him to overcome them but since an operation has to be faced only once or twice in his lifetime it is justifiable to take the course, less satisfactory in theory, of exploiting the sedative and euphoric effects of drugs.

### The Use of Sedative Drugs

The dose of premedicating drug a patient will require can often be roughly gauged by his resistance to alcohol. For example, the plethoric middle aged man who is accustomed to drink half a bottle of whisky a day will need a much larger dose of premedicating drug than will the frail woman as also will robust individuals, heavy cigarette smokers and for an unknown reason, malarial subjects.

Pre anæsthetic medication ranges from the administration of 5 gr of aspirin to the production of basal anæsthesia. The sedative drugs used for pre anæsthetic medication exert a beneficial effect by counteracting the factors which increase reflex irritability. Many of these drugs if exploited to the full would provide anæsthesia deep enough for minor surgery but for one reason or another it is inadvisable to use the large doses which would be necessary. This is well illustrated by morphia the specific effect of which in therapeutic doses e.g.  $\frac{1}{4}$  gr to an adult is to relieve pain. Although 1 gr of this administered to an average adult may produce anæsthesia deep enough for a minor operation it is unsuitable for use as a sole anæsthetic, because doses sufficient to establish surgical anæsthesia produce dangerous

Premedication in a case such as this is of great value since it minimises the effects caused by the adverse mental and physical conditions of the patient. The effect of reassurance alone does much to allay fear and this effect can be increased by sedative drugs such as the barbiturates or aspirin. Pain can be relieved by morphia or aspirin. The starting point of anæsthesia can by these means be reduced even below the level A say to X<sup>1</sup>.

The use of premedication spares the patient much mental anxiety shortens the induction period and makes it tranquil and diminishes the amount of anæsthetic required. Moreover post operative depression is less because less subsequent anæsthetic is needed and because the exhaustion which frequently follows the state of stimulation in the hyper-alert patient is eliminated.

It must not be overlooked that any condition which causes pain pyrexia or fear if continued to a degree which exhausts the sympathetic nervous system will result in shock a lowered metabolic rate and a commensurate depression of the starting-point of anæsthesia. This is well seen in the patient exhausted from exposure severe wounds or by infective disease of some months duration. Premedication with morphia or the barbiturates is here not necessary and is even contra indicated if the superimposition of further depression is considered dangerous.

### Psychological Premedication

Reduction of alertness which may be regarded as the beginning of anæsthesia can be accomplished in varying degree not only by drugs but by psychological treatment. Such treatment can often greatly reduce the increase in resistance to anæsthetics brought about by fear or pain. The method employed depends upon the anæsthetist and may range from an unimaginative slap on the back with a hearty

You'll be all right to the other extreme of hypnotisation. The time available often makes it impossible to prepare the patient psychologically as fully as is desirable but every opportunity to do so should be utilised.

A patient before an operation is in an abnormal frame of mind. It must be remembered that the operation is a crisis in his life vastly more important to him than it is to the anæsthetist or dentist for whom such an occasion is a matter of routine. Expectation that the operation will be painless does not necessarily remove a patient's dread some fear the anæsthetic more than the operation. The patient's attitude towards anæsthesia must not be judged by the experience of

anæsthetic itself have worn off, the slowly excreted premedicant drug keeps the patient unconscious although painful stimuli may cause extreme restlessness since pain is not obtunded. If there is no pain he will sleep peacefully. These two results are seen in the alternation between peaceful sleep and extreme restlessness when nembutal is used to produce amnesia of the pains of labour. The restlessness which occurs during recovery from a painful operation must be controlled by administration of morphia or a similar analgesic drug. It is noteworthy that a patient exposed to pain is more restless when under the influence of a cortical depressant drug than when conscious. Probably this is to be explained by loss of control of the higher centres enhancing the responses to pain and other stimuli—a condition existing in the decerebrate animal.

The basal anæsthetics (e.g. avertin, paraldehyde), the ultra short-acting barbiturates (e.g. thiopentone, hexobarbitone) and the inhalation anæsthetics, all produce unconsciousness comparatively early and recovery from anæsthesia is not unduly prolonged, although it is generally slow from the effects of drugs of the first two groups. Drugs of the last two groups abolish response to painful stimuli in doses which do not cause marked respiratory depression, but at the light level of unconsciousness which is all that should be aimed at with drugs of the first group, while respiration is not unduly depressed, response to painful stimuli is not abolished.

For the purposes of premedication, operations can be divided into those on ambulatory patients and those on hospitalised patients. The former are of necessity trivial.

## PRE-ANÆSTHETIC MEDICATION FOR THE AMBULATORY PATIENT

Most of the operations performed on ambulatory patients are dental extractions under nitrous oxide. In few branches of anæsthesia can premedication be more useful, and in no branch is it more neglected. The anæsthetist is asked to provide tranquil anæsthesia in a patient whom he may have met only a few minutes before. Since the patient is probably nervous and usually healthy, he will be to some degree anæsthetic resistant, but he will in any case expect a recovery rapid enough for him to go home soon after and even to continue with his usual day's routine.

There may not be an opportunity of arranging for premedication with sedative drugs in every case, and the anæsthetist should always

respiratory depression A therapeutic dose of  $\frac{1}{4}$  gr has hardly any obvious effect upon respiration but it is a quarter of the amount which might produce full anæsthesia and any anæsthetic will be effective in much lower dosage if morphia has been given beforehand This potential anæsthetic effect of morphia is often overlooked because the pre anæsthetic dose does not produce sleep

This facilitation of anæsthesia can best be demonstrated when the premedicating drug is followed by an anæsthetic injected intravenously, the quantity of which can be measured accurately If for example thiopentone 0.6 g is necessary to anæsthetise for a minor operation a nervous man in pain 0.5 g would probably suffice if his nervousness is reduced by psychological treatment, and if such treatment is reinforced by  $\frac{1}{4}$  gr morphia then thiopentone 0.3 g might well prove to be sufficient The practical importance of morphia in reducing the amount of anæsthetic subsequently necessary is best appreciated when a therapeutic dose of this drug is given before the administration of  $N_2O$  Although the patient may not be made sleepy not only is anæsthesia achieved and maintained with a lesser element of anoxæmia than is otherwise necessary but the combination will produce peaceful anæsthesia in cases where  $N_2O$  alone would be quite inadequate

Different sedatives have characteristic and widely different actions Some e.g. the short acting barbiturates act primarily by relieving fear and producing sleep others e.g. drugs of the opium group relieve pain when given in doses which do not produce sleep These differences can be attributed to selective action on different parts of the brain For example the cerebral cortex is specially sensitive to the short acting barbiturates e.g. nembutal and the midbrain to alkaloids of the opium group of which morphia is the most frequently used

The short acting barbiturates diminish apprehension and fear and in large doses produce drowsiness and unconsciousness long before they produce any notable change in the reflex response to pain They are quite unsuitable for the production of general anæsthesia since among other drawbacks elimination even after therapeutic doses is slow and after the large doses which would be necessary to produce general anæsthesia recovery of consciousness might be delayed for as long as forty eight hours If premedication is achieved solely by means of these cortical depressant drugs the patient benefits in that he will be asleep before the inhalation anæsthetic is given but the amount of the latter necessary to ensure lack of response to painful stimuli is but little reduced During recovery after the effects of the inhalation

to the average patient, should allow the dentist to accomplish any ordinary extractions. For convenience the methods by which  $N_2O$  anæsthesia can be facilitated are summarised.

**1 The patient's reflex irritability and resistance should be diminished by—**

(a) Gaining his confidence. Common fears and their treatment are dealt with on p. 180.

(b) Ensuring that he has a good sleep on the night before operation.

(c) Reducing apprehension by premedicating drugs such as aspirin, bromides or for alcoholics even by alcohol.

**2 The anæsthetic effect of the gas can be reinforced by—**

(a) Diminishing the oxygen intake.

(b) The preliminary use of a small dose of thiopentone given intravenously.

(c) Adding a more powerful agent such as ethyl chloride.

Whenever possible the dentist and anæsthetist should see that the measures enumerated under heading 1 are carried out in every case while those under 2 are resorted to only when necessary.

## PRE-ANÆSTHETIC MEDICATION FOR THE HOSPITALISED PATIENT

The desirability of giving some sort of pre-anæsthetic sedative drug before any operation cannot be disputed but the choice of drug and the dose in which it shall be administered is governed by such considerations as the desirability of long or short post-operative unconsciousness, the nursing facilities available, the length of time a patient is to be hospitalised and the accompanying financial implications.

**Morphia**—In the premedication of hospitalised patients morphia is more extensively used than any other sedative drug. Certain aspects of its action were dealt with earlier in this chapter. The standard dose for premedication is  $\frac{1}{10}$  gr per stone of body weight, with a maximum of  $\frac{1}{4}$  gr. For this purpose larger doses should rarely be given because they are accompanied by a depressant action on the respiratory centre. Although its anodyne effect is noticeable 10–15 minutes after hypodermic injection, maximum depression of respiration and of metabolism is not seen for a further hour. If morphia is given intravenously respiratory depression is maximal after about 10–15 minutes. When used for premedication morphia should be given sufficiently early for this maximum respiratory depression to



make an effort to gain the patient's confidence both immediately before and during induction. Even such brief psychological preparation makes it possible to achieve peaceful anæsthesia with a smaller amount of anæsthetic than would otherwise be necessary.

Ideally preparation for a dental extraction should begin with the administration of a sedative the night before the operation in order to prevent the irritability which follows loss of sleep. Even if a sedative should be unnecessary it will do no harm and it may do a great deal of good. Aspirin usually to hand in every household is suitable for this purpose. For an average adult the dose is 5-15 gr. Alternatives are phenobarbitone 1 gr. or potassium bromide 20 gr.

If the patient has to resume his normal occupation soon after recovery the choice of premedication to be given on the day of operation is restricted. It is neither necessary nor desirable to make him drowsy but the dose of premedicant drug should be sufficient to obviate anxiety at the thought of the anæsthetic. Two half doses of the premedicating drug used the night before should be given on the day of the operation. It is a good plan to give half the full dose at breakfast and the remaining half one hour before operation. These proportions, one full dose at night and two half doses, one at breakfast and the other one hour before the operation, should always be observed. Thus if an extraction is timed for midday aspirin 15 gr. should be taken on retiring the previous night.  $7\frac{1}{2}$  gr. is taken at breakfast time and again at 11 a.m.

When nitrous oxide without adequate premedication is used for a nervous patient, irritable and worn out by toothache, it may be necessary to diminish the oxygen percentage in the inhaled mixture to such a degree that asphyxial movements of the body make operating difficult. Post-operative collapse is then probable. One of the secrets of good nitrous oxide anæsthesia is correct premedication, for by this means undue asphyxia with its complications and sequelæ can be avoided. It should be noted that for anæsthesia by nitrous oxide premedication is not only a comfort to the patient but of considerable practical assistance to the anæsthetist. In fact it may determine the difference between a turbulent and a peaceful anæsthesia. In contrast when anæsthetics more powerful than nitrous oxide are used sedative premedication is given chiefly as a comfort to the patient. In this case although the amount of anæsthetic subsequently needed is indeed reduced the adequacy of the ultimate anæsthesia is not affected since the potency of the anæsthetic drug is such that any desired depth of anæsthesia can be produced.

Nitrous oxide given nasally by a moderately experienced anæsthetist

the elderly because it may produce delirium and restlessness, frequently shown by picking at the bedclothes. Between these age groups hyoscine will be found advantageous. In doses of  $\frac{1}{100}$  gr it is sedative but not soporific. It is not suitable, however, for the ambulatory patient for in many ways the patient under hyoscine behaves like one slightly concussed and not fully responsible. His actions are automatic and there is loss of memory for events following the injection. It is for this reason that it is so useful in childbirth. During the twilight sleep of hyoscine the patient complains and appears to feel the pains of labour but remembers little or nothing afterwards. Similarly when it is given as a premedicant drug before an operation although the patient may talk rationally and object to the inhalation anæsthetic, if one is given he seldom remembers the induction.

For hospitalised patients morphia may be conveniently combined with hyoscine in the proportion of 25 : 1 (e.g. morphia  $\frac{1}{4}$  gr hyoscine  $\frac{1}{100}$  gr) given  $1\frac{1}{2}$  hours before operation. By the time the patient reaches the operating theatre the sedative effect will be maximal. The summation of the actions of these two drugs causes a marked general depression of nervous activity without corresponding depression of the respiratory centre. Respiration, in fact, is little more depressed than when morphia alone is given. Besides the amnesic action of hyoscine its action on the parasympathetic nervous system is generally sufficient to make the use of atropine unnecessary.

**Alcohol**—Much confusion exists as to the position of alcohol in relation to anæsthesia. The narcotic effect of alcohol is seen daily yet oddly enough this action is seldom taken into account when anæsthesia is considered. Numerous cases are on record where the ingestion of a large quantity of alcohol has led to death with signs apart from the smell of the breath indistinguishable from those produced by an overdose of ether. Although its use as a basal or as a sole anæsthetic has now been abandoned it is well to remember that this drug did enjoy a brief popularity as an anæsthetic for a few years after Marin's thesis in 1929<sup>3</sup>. We have produced anæsthesia for abdominal and other operations solely by giving alcohol intravenously. Anæsthesia and recovery were entirely uneventful. There is however no method of administering alcohol which is simple enough to warrant its routine use.

We have often anæsthetised patients who had resorted freely to the whisky bottle to assuage the pains of toothache and have found them easy subjects for nitrous oxide. A drunken patient who has to be anæsthetised for say reduction of a fractured tibia will be

be manifest before the anæsthetic is begun. If this precaution is not taken, absorption of morphia will continue after anæsthesia has been achieved and dangerous depression of the respiratory centre may result.

If by mistake morphia is given in the standard premedicant dose before drugs such as avertin or cyclopropane which also have a marked respiratory depressant effect the course of the anæsthesia may be fraught with difficulty and danger. If respiratory depression becomes pronounced the patient will need undivided attention until it has worn off.

Although subsequent anæsthesia is eventually achieved with less anæsthetic than if morphia were not given full doses of this drug in the rare cases where ether is used for induction as well as for maintenance of anæsthesia have the paradoxical effect of making the attainment of deep anæsthesia difficult. Before the beginning of induction the respiratory depressant effect of the morphia may not be obvious but it becomes so immediately ether is used. The sensitivity of the respiratory tract is scarcely diminished by morphia so that the irritant effect of ether on the larynx raises the threshold of the respiratory centre to  $\text{CO}_2$  (p. 48) above the level to which it has already been raised by the morphia. Shallow and slow breathing results and if the anæsthetist attempts to shorten the induction period by increasing the concentration of ether vapour frank laryngeal spasm raises the threshold of the centre still further. During this period the blood ether will be absorbed by the body tissues and anæsthesia may even become lighter rather than deeper.

The special use of morphia as a premedicating drug for  $\text{N}_2\text{O}$  anæsthesia is well recognised and it is also of value before local anæsthesia because it reduces appreciation of any discomfort attendant on the operation but it is seldom used for the ambulatory patient in the dental surgery. It could and possibly should be used much more for these cases than it is at present. One marked disadvantage is that a patient under the influence of morphia should not be allowed to travel unaccompanied any more than he would be if mildly intoxicated by any other drug. Further liability to nausea renders a morphinised patient unfit to undertake his normal work for a few hours after injection.

Morphia is of great value in the recovery period to abolish restlessness from pain (p. 290).

**Hyoscine (scopolamine)**—This drug is frequently used in hospitalised patients because of the combination of its depressant action on the central nervous system and its effects in reducing mucus secretion. We have not used it for children and avoid giving it to

On many occasions we have anaesthetised patients alcoholic and otherwise who had indulged freely in alcohol to relieve toothache. The dentist has expected lack of co operation, but almost invariably we have found these patients easier to anaesthetise than if they had not been under the influence of alcohol.

With the great number of good pre anaesthetic drugs now at the anaesthetist's disposal we do not suggest that alcohol should be used to produce sedation, but we feel strongly that any patient particularly the alcoholic should not be deprived of his customary alcohol for the few days preceding an operation. Such deprivation greatly increases reflex irritability and resistance to the onset of unconsciousness.

There is no objection to a patient drinking a brandy and soda, or a whisky and soda one hour or even immediately before the anaesthetic proper provided it helps him to face the ordeal. In fact he will be all the better for it. On the other hand the patient who finds this necessary must be regarded with considerable suspicion, as he is likely to be accustomed to indulgence in alcohol and therefore to give trouble but such last-minute fortification will make him less rather than more troublesome.

## BASAL ANÆSTHESIA

As their name suggests basal anaesthetics are used to produce a basic level of unconsciousness which can be deepened to surgical anaesthesia by an inhalation anaesthetic. In larger doses they could be used as the sole means of producing surgical anaesthesia but such use is rarely warrantable.

Broadly speaking an anaesthetic may be considered safe only if it affords a satisfactory margin between the quantity necessary to provide good operating conditions and that which would cause respiratory failure and if it can be administered by a method which enables the anaesthetist to deepen or lighten anaesthesia at will. Although most of the sedative drugs in common use provide a wide safety margin it is only with inhalation anaesthetics that the depth of anaesthesia can be varied from moment to moment as may be demanded by fluctuation in the state of the patient and the exigencies of the operation. If an attempt is made to obtain full anaesthesia by a non inhalation anaesthetic an error of judgment may easily cause a lethal dose to be given. In any case the rate of recovery from any such drug is so slow that it should rarely be used as the sole anaesthetic for a long operation. Nevertheless non inhalation anaesthetics have a wide sphere of usefulness as basal anaesthetics.

found to need much less anæsthetic than if he had been sober. The effect of alcohol here is the same as that of small doses of ether and it is evident that if the patient is under the influence of small doses of ether he will need less subsequent anæsthetic than if he were not.

On the other hand alcoholism is an addiction and tolerance of and resistance to alcohol and other narcotics are soon acquired. A patient who drinks alcohol daily acquires a resistance to the effects of anæsthetics in the same way as if he inhaled daily small quantities of ether. For this reason the chronic alcoholic unless heavily premedicated or unless he is under the influence of alcohol, is very difficult to anæsthetise with a weak anæsthetic such as  $N_2O$ . Because resistance to anæsthesia is well known to follow the continuous taking of alcohol a regular drinker is frequently warned not to take alcohol for the few days before he is to be anæsthetised. Such advice is greatly mistaken. It rests on a failure to appreciate that resistance is produced by the adaptation of the body to alcohol and is not due to the effect of alcohol recently taken. It is the alcohol taken during the last ten years which causes the resistance but the alcohol taken during the last forty eight hours acts as a sedative and diminishes resistance. The intoxicated man is easy to anæsthetise whereas the chronic alcoholic deprived of alcohol is very difficult. The following examples in our own experience illustrate the typical effects of alcohol on anæsthesia.

A drunken man was brought to hospital with a broken leg and a dislocated shoulder. He was given  $N_2O$ . He did not struggle and a mixture with a high percentage of oxygen provided good muscular relaxation which allowed the shoulder to be reduced with ease. He was warded in hospital for two days and then anæsthetised for reduction of a fractured tibia. In the meantime he had been deprived of alcohol according to the hospital routine. Standard premedication proved insufficient. During induction by a competent and experienced resident he struggled violently and was not subdued until a large amount of anæsthetic had been given. Such struggling could have been largely averted by correct premedication or even by two or three strong whiskies and sodas in the few hours preceding induction.

In another case the patient was a woman whom the dentist stated had been an alcoholic but he expected that she would not cause any trouble since she had taken a cure and had been teetotal for six months. There was no reason to doubt the latter part of the story yet she proved difficult to manage showing reactions to anæsthesia typical of the chronic alcoholic.

will fall asleep again in a few minutes and the inhalation anæsthetic may then be commenced

Of the many basal anæsthetics available we discuss here only those of which we have had considerable practical experience. The dose of each drug mentioned is that for an adult patient in average health. This standard dose should be reduced for the æsthenic and increased for the robust or resistant patient

### The Rectal Route

Gwathmey in 1913 gave ether mixed with olive oil by rectum to a patient in bed before taking him to the operating theatre and as Rowbotham indicates this was the first big step towards preventing the mental distress which often has such a deleterious effect upon his condition before, during, and after operation.<sup>4</sup>

To obtain successful results from administration of anæsthetics by the rectum the following routine is recommended. An enema should be given the night before operation. Whether this has been done or not it is unnecessary and even undesirable to give an enema on the morning of operation, since this increases the sensitivity of the rectum for a few hours and the anæsthetic given may be expelled. The patient lies on his left side and two pillows are placed under his buttocks



FIG. 22.—This position by allowing the fluid to run freely into the bowel hastens absorption and prevents discomfort from distension of the rectum

Nothing is to be gained by running the fluid in slowly but it should not be run in fast enough to cause discomfort from rectal distension. With children particularly if they are unco-operative expulsion of the fluid can be prevented by pressing the buttocks together both during and after the rectal injection

There are many basal drugs at the anæsthetist's disposal. They can be given by rectum, by mouth or intravenously. In all cases the onset of sleep is peaceful and pleasant. For some reason for which no adequate explanation can be offered drugs given by mouth, rectum or subcutaneous or intravenous injection produce a more pleasant transition from consciousness to unconsciousness than do drugs which must be inhaled. With basal anæsthetics the onset of narcosis is almost imperceptible and so much resembles normal sleep that resistance to anæsthesia is not mobilised as often as by inhalation anæsthetics. The rate at which the patient becomes unconscious depends on the rate of absorption and is therefore greatest when the drug is given intravenously. The length of the recovery period depends on the drug chosen and the amount given.

Some writers have stated that the preliminary use of a basal anæsthetic increases the risk of post-operative morbidity which accompanies the administration of an inhalation anæsthetic. This view is erroneous although it is true that return to consciousness is usually delayed by a basal anæsthetic, so that the patient needs longer post-operative care.

The one tenable argument against most basal anæsthetics is that the prolonged period of unconsciousness which they produce increases the period pre- and post-operative during which the patient must not be left without skilled supervision. For this reason a shortage of nursing staff prevents the fuller use of basal premedication.

There are no age limits to the use of basal anæsthesia. In the very young metabolism is high so that a proportionately higher dose is necessary. The basal metabolic rate of the elderly is low, their dread of surgery is less than in the young adult and if a basal anæsthetic is to be given these factors make it necessary to reduce the dose accordingly. If this adjustment is not made deep and prolonged unconsciousness may result. This is particularly undesirable because protracted immobility in elderly patients is liable to be followed by hypostatic congestion and œdema of the lungs and by venous thrombosis which may result in pulmonary embolism.

The duration of sleep produced by the basal anæsthetics about to be described varies from a few minutes to several hours. Since the sleep produced will not be deep all stimuli should be eliminated as far as possible. The room should be quiet and the patient protected from strong light. He must not be jolted during transport to the theatre nor bumped clumsily on to the operating table. Under avertin narcosis a patient may be roused during transit to the theatre and may be apparently awake on arrival. If he is left undisturbed he

contra indications to its use are (1) the prolonged unconsciousness (2) its excretion in the breath makes the atmosphere of the nursing room objectionable, and (3) in our experience it has occasionally proved sufficiently irritating to the rectum to make its retention uncertain

**Thiopentone and hexobarbitone**—We sometimes administer these barbiturates by the rectal route in doses of 0.1 g per year of age, with a maximum of 2 g. The powder is dissolved in 20–30 c.c. of water and the solution run into the rectum. The patient is usually lightly asleep in 15 minutes and remains so for a further half-hour.

### The Oral Route

The advantage of this route is that it involves the minimum of fuss for everyone concerned. The disadvantage is that gastric and intestinal motility and absorption are notoriously unreliable in the nervous individual, the very one for whom basal anaesthesia is particularly desirable. The oral route is successful in about 95 per cent of cases but in the remainder the anaesthetist is faced with the problem of dealing with a patient who has ingested but not absorbed a strongly depressant drug. If the operation is postponed, all concerned are inconvenienced. If it is decided to proceed and an inhalation anaesthetic is given, the patient will have lost the advantages of basal anaesthesia and the anaesthetist will have to allow for absorption of the ingested drug at an unpredictable time during unconsciousness.

Some disappointments are inevitable when basal anaesthetics are given orally. It cannot be promised, as with a basal anaesthetic given rectally or intravenously, that sleep will certainly follow.

One of us was called in to anaesthetise a child in the early afternoon. She had been put to bed at this unusual time in a bedroom transformed into an operating theatre. She was apprehensive of course in such surroundings, even though she had not been told she was to have several teeth extracted. The family doctor insisted upon nembutal by mouth as the basal anaesthetic and this was given one hour before it was intended to start the operation. Since the drug had not produced any effect by the appointed time the operation was postponed. An hour later the child fell into a sleep which lasted several hours. If she had been premedicated with bromide for two or three days beforehand, she might have been spared her apprehensions and gastric absorption might not have been delayed. Alternatively she should not have been allowed to know anything of the preparations for operation and should have been given nembutal in place of lunch,



**Avertin fluid (Bromethol B P)** [Tri bromethyl alcohol ( $\text{CBr}_3\text{CH OH}$ ) in amylene hydrate] —The dose is 0.1 c c per kilo of body weight of the patient. The avertin fluid is shaken vigorously with forty times its volume of distilled water at approximately body temperature tested with Congo Red (to make sure that the solution is not acid enough to turn this blue) and then run into the rectum through a catheter. The patient rapidly becomes drowsy unconsciousness is reached in 10–30 minutes and lasts about an hour and a half. Since avertin has an appreciably depressant action on respiration the standard dose must be greatly reduced if another drug with this action e.g. morphia has been given within the previous 3 hours. One advantage of avertin very useful for jaw surgery is that the masseter muscle is relaxed even during light anæsthesia.

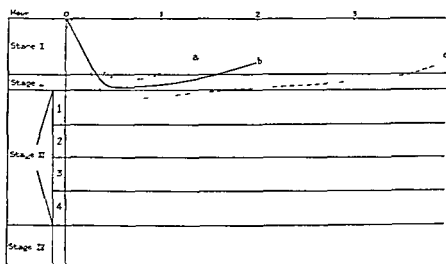


FIG. 23 —Chart of sleep resulting from rectal administration of (a) thiopentone (b) avertin and (c) paraldehyde in the doses mentioned in the paragraphs dealing with these drugs. There is no significant difference in the time taken to produce unconsciousness with all three but they differ in the depths of depression produced. Since the rates of excretion differ there is a corresponding difference in the duration of their effects.

**Paraldehyde ( $\text{CH}_3\text{CHO}$ )<sub>3</sub>** —The dose is 1 drachm per stone of body weight with a maximum dosage of 1 oz (8 drachms). Before being given by rectum it should be thoroughly shaken with twelve times its volume of water or saline or it can be given well mixed with an equal volume of olive oil. Unconsciousness comes on rather more slowly than after avertin and lasts longer—usually four to five hours. It does not produce any marked respiratory depression. The main

contra indications to its use are (1) the prolonged unconsciousness (2) its excretion in the breath makes the atmosphere of the nursing room objectionable, and (3) in our experience it has occasionally proved sufficiently irritating to the rectum to make its retention uncertain

**Thiopentone and hexobarbitone**—We sometimes administer these barbiturates by the rectal route in doses of 0.1 g per year of age with a maximum of 2 g. The powder is dissolved in 20–30 c.c. of water and the solution run into the rectum. The patient is usually lightly asleep in 15 minutes and remains so for a further half hour.

### The Oral Route

The advantage of this route is that it involves the minimum of fuss for everyone concerned. The disadvantage is that gastric and intestinal motility and absorption are notoriously unreliable in the nervous individual the very one for whom basal anaesthesia is particularly desirable. The oral route is successful in about 95 per cent of cases but in the remainder the anaesthetist is faced with the problem of dealing with a patient who has ingested but not absorbed a strongly depressant drug. If the operation is postponed all concerned are inconvenienced. If it is decided to proceed and an inhalation anaesthetic is given the patient will have lost the advantages of basal anaesthesia and the anaesthetist will have to allow for absorption of the ingested drug at an unpredictable time during unconsciousness.

Some disappointments are inevitable when basal anaesthetics are given orally. It cannot be promised as with a basal anaesthetic given rectally or intravenously that sleep will certainly follow.

One of us was called in to anaesthetise a child in the early afternoon. She had been put to bed at this unusual time in a bedroom transformed into an operating theatre. She was apprehensive of course in such surroundings even though she had not been told she was to have several teeth extracted. The family doctor insisted upon nembutal by mouth as the basal anaesthetic and this was given one hour before it was intended to start the operation. Since the drug had not produced any effect by the appointed time the operation was postponed. An hour later the child fell into a sleep which lasted several hours. If she had been premedicated with bromide for two or three days beforehand she might have been spared her apprehensions and gastric absorption might not have been delayed. Alternatively she should not have been allowed to know anything of the preparations for operation and should have been given nembutal in place of lunch.

and encouraged to lie down on the sofa while somebody read to her. Under these conditions the nembutal would have been absorbed and once asleep the child could have been undressed and put to bed.

The percentage of failures will be decreased by any preliminary procedure which reduces apprehension and ensures normal functioning of the stomach. The night before operation an adult should receive phenobarbitone 1 gr. to ensure a good night's sleep and 2 hours before operation morphia  $\frac{1}{8}$ – $\frac{1}{4}$  gr. subcutaneously.

**Nembutal**—This barbiturate is a bitter powder which is supplied in gelatin capsules. The dose for an adult is 3–6 gr. Unconsciousness comes on in about 20 minutes and lasts for about 1½ hours. The dose for children in *grains* (not grams) is 0.6 per stone of body weight with a maximum of 3 gr. Since it is often difficult to persuade a child to swallow a capsule the powder can be shaken into a medicine glass and its bitterness largely disguised by the addition of honey and orange juice.

### The Intravenous Route

The barbiturates which are given intravenously can be classified roughly as ultra short- and short acting according to the rates of their excretion.

**Thiopentone and hexobarbitone**—These belong to the ultra-short acting group and are discussed more fully on p. 138 ff. As basal

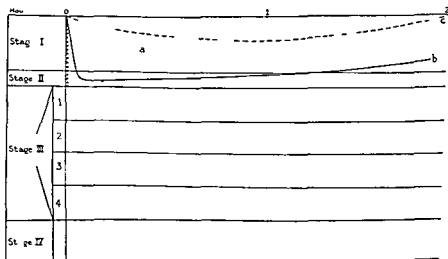


FIG. 24.—Chart of sedation produced by (a) intravenous thiopentone (b) intravenous nembutal and (c) subcutaneous morphia.

anæsthetics they can be used for the ambulatory patient if only a very small dose (e.g. 0.25 g) is given rapidly, to a patient in bed, double this dose or even more may be necessary to allow him to be transported in the unconscious state to the operating theatre.

**Nembutal**—Of the short acting barbiturates, we have had most experience with this drug which is a powder readily soluble in water. It must be injected slowly and the administration stopped when unconsciousness is reached. On an average this is attained by 3–6 gr of nembutal. It is excreted slowly. Average doses produce unconsciousness lasting for  $1\frac{1}{2}$  to 2 hours and drowsiness persists for many hours afterwards. The intravenous use of nembutal is now extremely rare as it has been superseded by thiopentone and hexobarbitone.

## ATROPINE

Although atropine is the only pre anæsthetic drug which does not have a sedative effect on the central nervous system it is used almost as a routine before general surgical operations. By depressing the activity of the parasympathetic nervous system which supplies secretory nerves to the salivary glands and to mucous glands of the bronchial tree atropine prevents the excessive secretion of mucus which otherwise occurs when the anæsthetic vapour is irritant. Hyoscine structurally related to atropine similarly depresses the parasympathetic nervous system. Only those who have given ether to unpremedicated patients realise fully the value of this protection both against obstruction of the airway by mucus and against post-operative chest complications.

Atropine is still recommended by some as a protection against sudden death in chloroform anæsthesia. Despite the work of Goodman Levy<sup>5</sup> it is still frequently taught that high concentrations of chloroform stimulate the vagus, and that the resulting inhibition of cardiac action is one of the causes of the sudden deaths which are known to occur during chloroform anæsthesia. It is probable that these deaths are due to ventricular fibrillation against which atropine offers no protection.

The dose of atropine for an adult is  $\frac{1}{100}$  gr and since it cannot be relied upon to be effective in smaller doses than  $\frac{1}{200}$  gr, this latter dose should be given even to an infant. Its action on the submaxillary gland becomes manifest after about 10 minutes. The cessation of salivary secretion will be found particularly helpful to surgeons operating in the mouth. To the patient however the dry mouth may be a cause of acute discomfort, particularly on a hot day. Atropine

and encouraged to lie down on the sofa while somebody read to her. Under these conditions the nembutal would have been absorbed, and once asleep the child could have been undressed and put to bed.

The percentage of failures will be decreased by any preliminary procedure which reduces apprehension and ensures normal functioning of the stomach. The night before operation an adult should receive phenobarbitone 1 gr. to ensure a good night's sleep and 2 hours before operation morphia  $\frac{1}{8}$ – $\frac{1}{4}$  gr. subcutaneously.

**Nembutal**—This barbiturate is a bitter powder which is supplied in gelatin capsules. The dose for an adult is 3–6 gr. Unconsciousness comes on in about 20 minutes and lasts for about 1½ hours. The dose for children in *grains* (not grams) is 0.6 per stone of body weight with a maximum of 3 gr. Since it is often difficult to persuade a child to swallow a capsule the powder can be shaken into a medicine glass and its bitterness largely disguised by the addition of honey and orange juice.

### The Intravenous Route

The barbiturates which are given intravenously can be classified roughly as ultra short- and short-acting according to the rates of their excretion.

**Thiopentone and hexobarbitone**—These belong to the ultra short acting group and are discussed more fully on p. 138 ff. As basal

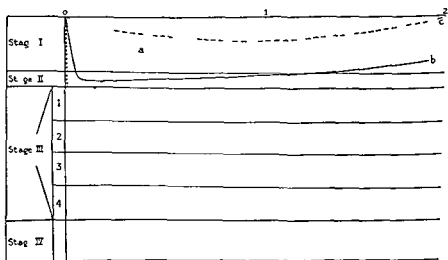


FIG. 24—Chart of sedation produced by (a) intravenous thiopentone (b) intravenous nembutal and (c) subcutaneous morphia.

## CHAPTER XI

### THE MORE DIFFICULT PATIENT

MENTAL and temperamental differences between individuals are as common as the more obvious physical differences, and they play an equally important part in affecting a patient's reactions to anæsthetics. The behaviour of an individual may differ so little from the average that, although the stress of fear and excitement temporarily reveals a previously unsuspected abnormality which makes him a difficult subject for anæsthesia yet he could hardly be described as abnormal. The anæsthetist should learn how to assess quickly the type of patient with which he has to deal so that he can foresee when anæsthesia is likely to be easy and when difficult. This is particularly important for the ambulatory patient whom the anæsthetist often sees for only a few minutes beforehand and who has usually had little or no premedication. The everyday social experience of a sympathetic observer will help him to detect such psychological traits as may be expected to influence the course of anæsthesia. The information he derives from the patient's behaviour during their short acquaintance before anæsthesia is begun together with his experience of the ways in which patients react during the early stages of anæsthesia enable him to foresee the probable behaviour of the patient during anæsthesia. The ability to 'size up' his fellow-men during a few minutes conversation is a valuable asset to an anæsthetist.

This chapter deals with the various categories of patients who present difficulties to the anæsthetist. The problems encountered and the principles employed in overcoming them are illustrated as far as possible by examples from anæsthesia in dental surgery.

### THE NERVOUS PATIENT

The way in which a patient reacts to the thought of an operation or of being rendered unconscious is of great practical importance to the anæsthetist. The fear reaction which is an essential factor in biological survival consists of a complicated series of changes which prepare an individual either to resist the incident which evokes fear, if necessary by means of physical violence or to run away. This is

should therefore not be injected an unnecessarily long time before the operation

There is no contra indication to the use of atropine in an ambulatory patient. There is no need for it, however, before simple extractions. If it is desired to ensure a dry mouth for a major jaw operation under local anæsthesia in the dental chair the drug can be given as a tablet by mouth half an hour before the operation.

#### REFERENCES

- (1) Shaw G B 1906 Preface to *The Doctor's Dilemma* London
- (2) Guedel A E 1937 *Inhalation Anesthesia* New York 61
- (3) Constantin J D 1929 *Lancet* **1** 1247
- (4) Rowbotham E S 1931 *Brit med J* **2**, 693
- (5) Levy A G 1922 *Chloroform Anæsthesia* London

The anaesthetist should treat with understanding reassurance, and encouragement any particular fear which he may detect. The fear most commonly confessed is that of losing consciousness. This vague fear occurs independently of and is much more frequent than fear of death. The patient may be afraid of losing self control or of behaving badly or of talking indiscreetly. If previously he has had a feeling of suffocation or a vivid 'nightmare' or any other unpleasant experience associated with anaesthesia he fears with a certain amount of justification that this experience is likely to be repeated.

Fear that too small a dose of anaesthetic will be administered, and that the pain of the operation will be felt, is far commoner than fear of over dosage. The patient who is afraid that the operator will begin work before he is unconscious often tries to keep his eyes open or holds up his hand or moves his arm up and down or attempts to speak or to repeat a monotonous sound to signify that he is still awake.

It is unusual for the patient to start to make any such movement or noise until he has taken about three or four breaths of gas. The administrator should take his cue immediately from any of these signs and put the patient's mind at rest by patting him reassuringly on the shoulder and by saying in a decided tone 'No you needn't worry. I know you are not nearly asleep yet. I promise you that

Mr Jones won't begin until you are completely asleep. Now I want you to breathe a trifle more deeply for a few breaths—one two excellent

One can often reassure the patient who indicates that he is not yet asleep by saying 'I am purposely putting you to sleep very slowly and giving you a lot of oxygen as a stimulant. That means that you will go off to sleep slowly but you will feel all the better afterwards. Although  $O_2$  is not in fact added until anaesthesia is established such a statement calms the patient and so conduces to a good recovery. A regular movement of the hand, arm or foot such as has been described when once initiated during consciousness may persist after unconsciousness has been reached but will disappear during deep anaesthesia. Sometimes merely putting the patient's arm down by his side will arrest these automatic arm movements.

Most people keep their eyes closed during the induction of anaesthesia. Nervous patients however tend to keep them open. For the sake of these the wise dentist will stand where he can be seen in front of the patient and endeavour to give the impression that he has no immediate intention of beginning to operate. He should show himself occupied for example in the contemplation of an X-ray film



aply described as the fight or flight mechanism, a primitive defensive mechanism of the body put into action as a result of stimulation of the sympathetic nervous system. A similar response is shown after the injection of small doses of adrenaline. There is increased activity in the relevant bodily functions. Dilatation of the pupil improves vision. the respiratory centre is rendered more sensitive facilitating intake of oxygen and excretion of  $\text{CO}_2$  and respiratory exchange is further improved by the substitution of oral for nasal breathing. liver glycogen is mobilised and the blood sugar thereby increased to supply muscles with energy. cardiac output is increased and blood pressure raised for the same reason. Reflex action time is shortened and mental alertness is increased and the ability to sleep correspondingly diminished to cope with the emergency. This means that the starting point of anæsthesia is raised considerably above the level at which consciousness is lost. that induction will therefore take longer, and that there will be more time and opportunity for the patient to behave violently before surgical anæsthesia is attained.

The highly nervous individual is one who readily shows this reaction to fear. He over-responds to ordinary stimuli and the mere thought of visiting the dentist is enough to upset him. Unless he has been calmed by appropriate premedication his inability to co-operate is liable to lead to difficulty in the induction period. It must not be thought that this over reactive type is always easily recognisable. Education and training can so modify a patient's reactions that his tendency to over respond may be hidden successfully until it is revealed by removal of higher conscious control as during induction of anæsthesia. Other modifying factors have a temporary effect and act in the ways one would expect. For instance if a patient who does not normally over react has been overworked or if he is upset by business or family worries he will be converted temporarily into the over reactive type and will behave accordingly. A familiar illustration is seen in hospital practice during a morning session of extractions when one sometimes has to deal with a patient who has just been on night duty. Such a patient is almost invariably difficult to anæsthetise. Similarly over reaction is seen in patients who have been kept awake all night by toothache.

Some degree of fear associated with the prospect of losing consciousness and of an operation is so natural as to be quite normal. Such nervousness is one of the commonest causes of difficulty during induction and in extreme cases is so marked that the patient is incapable of co operating with the anæsthetist.

Thus a man may, by years of practice have developed so perfect a voluntary restraint that his friends do not realise that he is a highly 'nervous' individual, that is his own secret. He is often one of the community's best types, intelligent and acutely alert to every danger, but with all his fears controlled and hidden.

The duration and intensity of the training process determine the extent to which a nervous patient is able to suppress the signs of fearfulness. The stiff upper lip training begins very early to modify the fear response. A good example is that of the young midshipman, who although acutely terrified by his visit to the dentist conceals his fear completely. However during induction of anæsthesia, voluntary control is the first function to be lost and then his real nervousness becomes manifest. The stage at which this occurs depends upon the strength of his initial self control. The young midshipman who has only relatively recently acquired the ability to control his emotions continues to obey instructions implicitly until the onset of unconsciousness. He thus retains his control for a longer time than does the patient who shows panic early in induction but patients with more rigorous self control retain it until various later stages of unconsciousness. In some patients it is so strong that it persists until late in anæsthesia by which time muscular and obvious mental action are in abeyance so that its loss is unnoticed and the anæsthesia remains smooth throughout.

The anæsthetist may be surprised by the patient whose emotional control is only superficial if he has not learned to be suspicious of the person who embarks on anæsthesia in an abnormally light hearted fashion for such light heartedness may represent an attempt to conceal a nervousness which is extreme. The patient at first co-operates perfectly but just as consciousness is lost he suddenly struggles violently and may get out of hand unless the anæsthetist has been anticipating this emergency and is ready to cope with it.

With many nervous patients difficulties are experienced only during the induction period and for the first few minutes of the supervening anæsthesia. The energy for resistance then seems to be dissipated and subsequent anæsthesia is easy. Loss of control is evidenced by lack of co-operation, the onset of vigorous oral breathing by movements of the arms and legs and sometimes by screaming. The patient may retch and spit out the prop and at last be subdued only with difficulty. When these reactions occur they indicate that fear influences the mind to such an extent that the subject is not susceptible to any reassurance. It is useless then for the anæsthetist to show impatience since this only increases the fear. These patients, once

or merely in gazing out of the window. If the patient should open his eyes before consciousness is lost he will then have less reason to suppose that the operation will commence prematurely than if he finds the dentist forceps in hand standing eagerly over him.

Fear that anæsthesia will be too great a strain for the heart may be indicated by the patient putting his hand to his heart during induction. The anæsthetist should pat him on the shoulder and inform him that the forceful beating is a usual indeed a reassuring phenomenon.

I know you can feel your heart beating strongly—that's nothing to worry about in fact it is a good sign. I want you to continue taking nice deep breaths exactly as you are doing now.

The nature of the proposed operation may itself determine the appearance of the fear reaction. A woman in particular is nervous and depressed or hysterical if she is to lose her front teeth. She may be afraid of wearing dentures or afraid that the need for this operation indicates the onset of old age and on such an occasion one may anticipate that she will not be as co-operative during induction nor as placid on recovery as if a molar tooth were to be extracted.

Fear is a complex phenomenon and patients often reveal specific individual fears. The person who is generally recognised by his friends as a nervous individual does not always present difficulties but sometimes the ordinarily well balanced individual shows a surprising instability when confronted with the prospect of being anæsthetised.

A striking instance of this contrast was seen one morning in two consecutive patients. The first a V.C. of the Great War entered the dental surgery for simple extractions in such a state of terror at the prospect of losing consciousness that it was thought advisable to postpone the operation and to arrange for its performance in a nursing home where he could be given a basal anæsthetic before nitrous oxide. The next patient a frail old lady nervous of crossing the road had no fear of unconsciousness or of an operation and anæsthesia with nasal nitrous oxide was quite smooth.

The anæsthetist is forewarned by an apprehensiveness which declares itself before the anæsthetic begins but an important degree of nervousness may exist without obvious signs leading to difficulties in the induction period which may take him unawares. It should be remembered that the reaction to fear varies in different individuals. Although it is governed by the laws of inheritance it may be brought under control by training or by the habits of a lifetime and as pointed out above it is not always possible to recognise when this has occurred.

metabolic rate depressed so that anæsthesia is started from the same level as on the first occasion, the original concentration of anæsthetic will then have its previous effect. It is however, of greater importance particularly for a nervous patient, to plan the dental work to be done so that as few separate anæsthetics as possible are needed. Although he feels less well after a long than after a short anæsthetic the average patient prefers one or two long anæsthetics during which the work can be completed than to have his visits to the dentist multiplied. If two visits are necessary it is generally preferable to do the less difficult extractions first so that after anæsthesia as short as possible and recovery as pleasant as possible the patient will not approach the second occasion with dread. However if the dentist is concerned about the operative difficulties he may begin with the more difficult extractions if he finds them even more formidable than he anticipated it is best for him to stop operating as soon as possible in order that post-operative depression may be minimal and to arrange for the patient to enter a nursing home for the rest of the work to be done at a single session.

## THE ANÆSTHETIC-RESISTANT PATIENT

There are two chief variables which determine an infinite variety of reactions to anæsthetics. These are the potency of the anæsthetic agent used and the resistance of the patient to the onset of unconsciousness. For instance if an average dose of a potent anæsthetic is administered to a patient depressed by shock, dangerously deep anæsthesia may be produced. On the other hand often in the dental surgery the anæsthetist is tempted to try to subdue a resistant patient with N<sub>2</sub>O the weakest of anæsthetic agents with the expectation that the resulting anæsthesia will be tranquil enough to allow difficult extractions to be accomplished. Only too often when the anæsthetist is inexperienced have hopes of this kind together with some of the surgical equipment been dashed to the ground!

The nature of the resistance shown by the anæsthetic resistant patient is different from that of the patient who is merely nervous. The latter is unco-operative during induction but is subsequently easy to manage. The former possesses a fundamental resistance to the effects of anæsthetics a resistance which is particularly evident when a weak anæsthetic such as N<sub>2</sub>O is used. It is with the healthy robust patient that anæsthetic difficulties are encountered when N<sub>2</sub>O is used—not as is generally supposed with the frail, elderly patient. Whilst

anæsthetised become peaceful and easy' others the wiry aggressive or resistant, will respond vigorously if stimulated at any stage during light anæsthesia

Once the fear reaction has occurred it is liable to recur if anæsthesia is allowed to lighten unduly. Moreover in an extremely nervous and resistant patient even deep  $N_2O$  anæsthesia sometimes so deep that asphyxial jactitation is imminent may be insufficient to prevent the operative stimulus evoking reflex muscular responses. Continuance of the stimulus may rouse the patient to mouth breathing and extreme restlessness and the operation must then be interrupted until anæsthesia is deepened again. As stated above the fear response is probably an adrenaline release mechanism producing a degree of alertness which is antagonistic to relaxation and sleep and therefore to the onset and maintenance of anæsthesia. Clinical experience shows that the antagonistic effect of the adrenaline mobilised by the fear reaction is so strong that in order to render the patient quiescent after the reaction has occurred and to restore nose breathing and a state of unresponsiveness to stimuli the  $O_2$  content of a  $N O O_2$  mixture may have to be reduced temporarily to a level considerably lower than that required to maintain satisfactory anæsthesia in a non nervous patient. Indeed the largest concentration of  $O_2$  in the mixture which will permit of successful anæsthesia can be regarded as an inverse measure of the amount of adrenaline in the circulation. Premedication (Chapter V) is the method *par excellence* by which the condition of a nervous patient can be so modified that he can be anæsthetised by nitrous oxide without resorting to extreme deprivation of oxygen.

### Cumulative Dislike of Anæsthetics

The resistance of an individual to the onset of anæsthesia may vary from day to day. It is a curious psychological phenomenon that during the course of five or six administrations the patient becomes increasingly unwilling to resign himself to unconsciousness brought about by *inhalation* anæsthetics however pleasant his previous experiences may have been. At the first or second administration of  $N_2O$  his tranquil state of mind may make it possible to give a relatively high percentage of oxygen. If the patient is more nervous on the next occasion this percentage has to be reduced in order to achieve the same depth of anæsthesia a phenomenon described by Hewitt as progressive intolerance to nitrous oxide. For this reason a patient who is at first normal may after repeated anæsthetics become a nervous patient. By reassurance and premedication the nervous element can be reduced and the patient's

enced in inducing anaesthesia with ether in such a patient because its irritating effects on the mucous membranes make it impossible to hasten the patient through the delirium stage. Struggling is then inevitable but once anaesthesia has been produced maintenance presents no difficulties because of the potency of ether. Although unconsciousness is rapidly produced with  $N_2O$  this agent alone is not strong enough to produce peaceful anaesthesia in a resistant subject unless a noticeable element of asphyxia is introduced. By the use of the more modern, powerful and pleasant anaesthetics (thiopentone cyclopropane, etc.) the anaesthetic state can be reached quickly and maintained easily without struggling. Resistance which would be obvious with  $N_2O$  is not seen with thiopentone where the stage of potential delirium is passed through in a few seconds and where the degree of resistance encountered is conveyed not in terms of a struggle but in terms of the dose of anaesthetic needed to produce peaceful anaesthesia. Thus an anaesthetic-resistant patient might need as much as 0.7 g. of thiopentone for an operation which in the average patient could be accomplished with 0.4 g. With a drug of rapid action such as thiopentone the alcoholic or other resistant patient is scarcely more difficult to anaesthetise than the average since all the difficulties can be overcome by administering a large dose.

FIG 25

If it is recognised before anaesthesia is begun that a patient is likely to be resistant a strap should be used so that the pelvis is fixed well back in the chair (p. 207). Without attracting the patient's attention the foot rest should be taken away. If the foot rest cannot be removed the feet should be placed on either side of the chair.



with the frail anæsthesia is easy throughout with the robust both induction and maintenance are difficult. There is only a narrow range of safe anæsthesia between marked response to an operative stimulus and asphyxial jactitation. Nervousness of course increases the difficulties encountered with this group of patients.

The anæsthetic resistant patient should be recognised as a definite type and dealt with appropriately. He possesses the quality of sthenicity (Gr *σθενος* = strength) a term used by American writers to signify excessive vital energy. Typical examples are the hunting major, the international footballer, the navy and the man who does not know when he is beaten. With a little experience resistance to the effects of anæsthetics can be estimated with fair accuracy. A direct comparison can be made with the resistance to the onset of unconsciousness from any other cause. A man who can take a severe blow and continue fighting will be anæsthetic resistant, so will the man who can drink half a dozen strong cocktails without showing any effects. If a patient is resistant to one anæsthetic he will be resistant to all. This state of resistance to unconsciousness may be innate or acquired. The determined wiry physically aggressive pugnacious individual is innately resistant while the patient who faints easily is not. Those who have acquired resistance include the physically fit robust subject in training and the alcoholic who by daily consumption of this drug has become resistant to the effects not only of alcohol but of all narcotics.

When an anæsthetic resistant patient presents himself for dental extractions he often gives a history of previous troublesomeness during anæsthesia. He is usually somewhat proud of this and does not spare any opportunity of telling of his previous difficult behaviour. Attention should be paid to the patient's warning that he takes gas badly. Wayward behaviour under  $N_2O$  is accounted for more often by the patient's idiosyncrasies than by unskilled anæsthesia. Every anæsthetist remembers with regret some case in which he did not regard the patient's warning, laying the fault in his own mind at the door of the previous administrator. Even if the patient is being anæsthetised for the first time or if he does not mention the difficulties of former occasions the anæsthetist with any experience can generally recognise that he is likely to prove resistant. While resistance to anæsthesia is most apparent when  $N_2O$  is used it will be seen also if ether is used as the sole anæsthetic for a major operation. With ether and  $NO$  in the resistant patient struggling is common with the former during induction with the latter during maintenance. Difficulties are experi-

the use of supplementary ethyl chloride. Smooth anæsthesia can then be maintained without reducing the oxygen content of the anæsthetic mixture to a dangerous degree. If it is decided to use a barbiturate intravenously the amount to be given depends on the degree of resistance anticipated from the patient and on the interval at which he has afterwards to leave the surgery in a normal condition.

If an anæsthetic resistant patient is not recognised as such before anæsthesia is begun and is given nitrous oxide he will cause difficulty. Even when cyanotic and obviously unconscious he is far from peaceful even though no operative stimulus is applied. He may purse his lips, roll his tongue, spit out the sponge, hold his head in an awkward position, be restless in the chair and hold his arms rigidly extended. Respiration is irregular, the breath being held in inspiration, and expiration is preceded by a grunt. In this type of case any of the following three procedures will be found useful.

1 The nose-piece is kept in position and a corner of the towel over the chest is turned up over the mouth and ethyl chloride is sprayed on to this (fig 42 p 162).

2 As soon as the patient is unconscious an ordinary marine sponge soaked in ether is put into the mouth and the mouth-piece is applied. This method is much more effective and pleasant than the description suggests. The effect, considering the amount of ether, is quite extraordinary. Very quickly the patient quietsens and anæsthesia remains tranquil. Recovery is unexpectedly rapid. In our experience this manoeuvre has not been followed by nausea or vomiting and the patient would appear to be unaware that ether has been given.

3 In a similar way a 3 c.c. phial of vinesthene may be poured on to the upturned bib or on to a marine sponge which is inserted into the mouth.

For the inexperienced anæsthetist the addition of ether will be found more fool proof and safer than the addition of ethyl chloride because the latter drug, if given in excess, rapidly produces dangerously deep anæsthesia. The after effects of the addition of ether, ethyl chloride or vinesthene are practically non-existent if their use is confined to the type of case for which they are necessary. If however these agents, particularly ethyl chloride, are used for the normal, or the frail patient, collapse may occur and return to consciousness be associated with nausea and vomiting.

Deepening of anæsthesia may be obtained by compressing the carotid arteries against the transverse processes of the cervical



Oxygen lack and surgical stimulus almost invariably cause some muscular response in these patients which may be violent enough to make them assume a position of opisthotonos unless these pre



FIG 26

cautions have been taken. If the foot rest is left in position when the strap is in use a powerful patient may press on it with sufficient force to break it.

Any movable furniture within reach of the patient's arms or legs should be moved out of range. Violence of an extent to justify this precaution is extremely uncommon but when it does occur it usually develops suddenly. Besides the genuine danger that the patient may damage himself because the attendants cannot control him and *incidentally the danger of his damaging the attendants the effect of his struggles may be disastrous to furniture within his reach.*

In addition to these preliminary precautions it is necessary when nitrous oxide is the anæsthetic to enhance its action by suitable means. As pointed out elsewhere the range of anæsthesia with  $N_2O$  is extremely narrow. During light anæsthesia a surgical stimulus will cause reflex responses—restlessness, struggling, mouth breathing and return to consciousness. If an attempt is made to provide deep anæsthesia by further diminishing the  $O_2$  intake asphyxial movements soon follow and later respiratory arrest. If  $N_2O$  is to be used for the unpremedicated anæsthetic resistant patient satisfactory results can be obtained by increasing the range of anæsthesia by a small dose of thiopentone or hexobarbitone given intravenously immediately before the  $N_2O$  or by

where the operation is to be performed on the pelvic organs on the thyroid gland or breast, it should unless urgent, be postponed during menstruation. Bleeding which is freer than normal occurs at this time in these areas from generalised pelvic congestion in the first group of operations, and in the second and third groups it is thought that there is increased vascularity of the organs concerned during the menstrual period as a result of some endocrine inter-relationship. In other cases, fear that there may be increased bleeding e.g. from the tooth sockets after dental extraction is without foundation since the rate of clotting of the blood is not slowed.

Not infrequently a patient volunteers that menstruation has begun and asks if the operation should be postponed on that account. Except in the instances mentioned she can be assured that this will not influence the operation in any way and with this knowledge the average patient decides to undergo the operation as arranged.

If, however, the patient is physically upset by menstruation or if there is any marked temperamental disturbance, it may be desirable to postpone the operation.

## PREGNANCY

Experience in anæsthetising patients from antenatal clinics leads us to believe that there is no evidence that administration of an anæsthetic to a pregnant woman renders her more liable to abortion or is harmful to the foetus. Since however there appears to be a greater tendency to spontaneous abortion at the times when the first three monthly periods would otherwise be due these times should be avoided for operations which are not urgent.

The choice of anæsthetic is determined by the patient's general condition and not by the fact that she is pregnant. If  $N_2O$  is used the foetus will not suffer from oxygen lack as much as is generally supposed because foetal hæmoglobin is saturated at a much lower oxygen tension than is that of the mother.

## PULMONARY DISEASE

If an extraction is necessary for a patient suffering from acute bronchitis the merits of local anæsthesia must be considered. If a general anæsthetic is decided upon nitrous oxide may be administered without any fear that its use will lead to an extension of the catarrh. Sedation with a full dose of morphia (say  $\frac{1}{4}$  gr. to a 10 stone adult) one hour before the anæsthetic makes it possible to produce adequate anæsthesia with less  $O_2$  deprivation than would otherwise be necessary.

vertebræ This method of producing temporary unconsciousness is well recognised by the Japanese and we have on occasions found it very helpful in increasing the effect of  $N_2O$  in short operations when this gas alone was not adequate

## ALCOHOLISM

The relation of alcohol and of the alcoholic patient to anæsthesia is dealt with on p 97

## MORPHIA AND COCAINE ADDICTION

We have anæsthetised both morphia and cocaine addicts and found them no more difficult than normal patients provided they had not been deprived of their customary allowance of these drugs

On one occasion we were asked to give nitrous oxide to a cocaine addict for dental extraction The patient's doctor under the impression that it would facilitate the anæsthetist's work diminished for 24 hours the patient's customary allowance of cocaine On arrival at the dental surgery the patient was restless and unco operative It would have been impossible to give him gas without resorting to forcible restraint He was taken home and brought back the next day having been given slightly more than his usual amount of cocaine He was then contented relaxed and co operative and as such was an easy subject to anæsthetise

An operation takes place only a few times in a patient's lifetime and everything possible must be done on these occasions to make him feel at ease If he happens to be a drug addict of any sort—cocaine, morphia alcohol or tobacco—he should not be deprived of the one essential he needs to face the everyday difficulties of life An increased allowance of his drug on this special occasion will soothe him and make the situation easy both for him and for the anæsthetist Premedication should take the form of a slightly larger dose of the drug on which the patient leans for support

## MENSTRUATION

There is no justification for the common impression that a patient who is menstruating should not be subjected to anæsthesia or to most operations on the grounds that it would be more detrimental to her than at any other time Surgeons appear to agree however that

where the operation is to be performed on the pelvic organs on the thyroid gland or breast, it should unless urgent, be postponed during menstruation. Bleeding which is freer than normal occurs at this time in these areas, from generalised pelvic congestion in the first group of operations and in the second and third groups it is thought that there is increased vascularity of the organs concerned during the menstrual period as a result of some endocrine inter-relationship. In other cases fear that there may be increased bleeding e.g. from the tooth sockets after dental extraction is without foundation since the rate of clotting of the blood is not slowed.

Not infrequently a patient volunteers that menstruation has begun and asks if the operation should be postponed on that account. Except in the instances mentioned she can be assured that this will not influence the operation in any way, and with this knowledge the average patient decides to undergo the operation as arranged.

If however the patient is physically upset by menstruation or if there is any marked temperamental disturbance it may be desirable to postpone the operation.

## PREGNANCY

Experience in anæsthetising patients from antenatal clinics leads us to believe that there is no evidence that administration of an anæsthetic to a pregnant woman renders her more liable to abortion or is harmful to the foetus. Since however there appears to be a greater tendency to spontaneous abortion at the times when the first three monthly periods would otherwise be due these times should be avoided for operations which are not urgent.

The choice of anæsthetic is determined by the patient's general condition and not by the fact that she is pregnant. If  $N_2O$  is used the foetus will not suffer from oxygen-lack as much as is generally supposed because foetal hæmoglobin is saturated at a much lower oxygen tension than is that of the mother.

## PULMONARY DISEASE

If an extraction is necessary for a patient suffering from acute bronchitis the merits of local anæsthesia must be considered. If a general anæsthetic is decided upon nitrous oxide may be administered without any fear that its use will lead to an extension of the catarrh. Sedation with a full dose of morphia (say  $\frac{1}{4}$  gr. to a 10 stone adult) one hour before the anæsthetic makes it possible to produce adequate anæsthesia with less  $O_2$  deprivation than would otherwise be necessary.

vertebræ This method of producing temporary unconsciousness is well recognised by the Japanese and we have on occasions found it very helpful in increasing the effect of  $N_2O$  in short operations when this gas alone was not adequate

## ALCOHOLISM

The relation of alcohol and of the alcoholic patient to anæsthesia is dealt with on p 97

## MORPHIA AND COCAINE ADDICTION

We have anæsthetised both morphia and cocaine addicts and found them no more difficult than normal patients provided they had not been deprived of their customary allowance of these drugs

On one occasion we were asked to give nitrous oxide to a cocaine addict for dental extraction The patient's doctor under the impression that it would facilitate the anæsthetist's work diminished for 24 hours the patient's customary allowance of cocaine On arrival at the dental surgery the patient was restless and unco operative It would have been impossible to give him gas without resorting to forcible restraint He was taken home and brought back the next day having been given slightly more than his usual amount of cocaine He was then contented, relaxed and co operative and as such was an easy subject to anæsthetise

An operation takes place only a few times in a patient's lifetime and everything possible must be done on these occasions to make him feel at ease If he happens to be a drug addict of any sort—cocaine morphia alcohol or tobacco—he should not be deprived of the one essential he needs to face the everyday difficulties of life An increased allowance of his drug on this special occasion will soothe him and make the situation easy both for him and for the anæsthetist Premedication should take the form of a slightly larger dose of the drug on which the patient leans for support

## MENSTRUATION

There is no justification for the common impression that a patient who is menstruating should not be subjected to anæsthesia or to most operations on the grounds that it would be more detrimental to her than at any other time Surgeons appear to agree however that

of the cyanotic patient with a strongly beating heart. Unless  $O_2$  is given the colour changes to grey and rapidly becomes ashen. Respiratory effort also fails so that if  $O_2$  is to be effective it must be given rapidly. Besides these colour changes any change in rhythm and volume of the pulse can be appreciated if the anaesthetist accustoms himself to keeping one finger on the pulse at the facial temporal or carotid arteries.

### Hyperpiesia

Most anaesthetics cause some fall of blood pressure. This is particularly noticeable with intravenous barbiturates and avertin. With nitrous oxide a slight rise occurs in the early stages if an asphyxial element is introduced. Profound asphyxia results in a fall of blood-pressure which is quickly corrected by the administration of oxygen. The early rise of blood pressure in nitrous oxide anaesthesia has led to an impression that this gas is contra-indicated for patients with high blood pressure. We are satisfied from practical experience in many thousands of cases managed by ourselves and hospital colleagues, that no harm has followed the use of nitrous oxide anaesthesia although many of the patients are known to have had high blood pressures. Nevertheless in dealing with a hyperpiesic patient any factor which may raise the blood-pressure further should be avoided as far as possible.

### ANÆMIA

(See also pp 56 and 74)

Patients suffering from anaemia, shock, or long-continued sepsis may be considered together. Although discussed in this chapter in reality they should be grouped under the heading the more easy patient. Compared with the doses one is accustomed to administering to normal patients appreciably smaller doses of any anaesthetic will produce satisfactory and often deep anaesthesia in these patients who are susceptible to any anaesthetic and as such are suitable subjects for nitrous oxide. Any difficulties which may arise during nitrous oxide anaesthesia are created by the anaesthetist expecting from them the responses usually shown by normal patients.

### PATIENTS CONFINED TO BED

When for dental extractions it is necessary to anaesthetise a patient in bed certain additional difficulties have to be overcome. To be accessible to the dentist the patient should be moved to the edge of

Since  $N_2O$  has no irritant action on the mucous membranes it can be used in phthisis or chronic bronchitis. If it is desired to avoid deprivation of  $O_2$  the action of  $N_2O$  may be reinforced by an intravenous barbiturate morphia or avertin. Alternatively anæsthesia can be produced by cyclopropane or by an intravenous barbiturate given alone. A warning is usually given against using ether in these conditions. This may be correct but it is doubtful if ether properly given through a perfectly clear airway to a patient premedicated with atropine is as undesirable as is usually suggested.

Any asthmatical subject whether in an attack or not should be liberally premedicated with morphia. Even in a normal patient the use of the barbiturates may be followed by a narrowing of the bronchial and bronchiolar lumina. We have seen a case of acute œdema of the lungs following a few hours after the administration of a barbiturate as the sole anæsthetic for an appendicectomy in a youth whom we had not known to be an asthmatic subject. Postural drainage and periodical suction through an endotracheal tube were instituted early and recovery followed.

### CARDIAC FAILURE

*The patient with an inadequate circulation due to cardiac failure must not be subjected to anoxia.* The mere presence of abnormal sounds of valvular origin is of little importance. What matters is the efficiency of the heart muscle. A heart may hypertrophy and compensate for a valvular lesion whilst a patient recuperating from a debilitating illness such as influenza may have a grossly impaired cardiac musculature of which no evidence can be found with the stethoscope. The cardiac musculature of a normal individual is well able to withstand a surprising degree of anoxia but the intolerance of the failing or the flabby heart even to a very small reduction of the oxygen intake is equally striking. If nitrous oxide is used for patients with inadequate circulation the oxygen content of the mixture must *not* be reduced below that in atmospheric air. The anæsthetic effect of this weak  $N_2O$   $O_2$  mixture can be reinforced by premedication with morphia or with thiopentone or by simultaneous administration of ethyl chloride in small doses. Ether is not contra indicated in fact if a long or deep anæsthesia is required it is the anæsthetic of choice since it can be administered with a high percentage of oxygen.

Occasionally  $N_2O$  may be given to a patient in whom degeneration of the myocardium has not been suspected or recognised. When the  $O_2$  content of the inspired mixture is reduced the blueness of the cheeks takes on a waxy appearance. The colour lacks the lividity

thiopentone or avertin, so that the face mask or nose piece is applied only after the patient has become unconscious

### PATIENTS WITH CLEFT PALATE

Patients with cleft palate present no special anæsthetic difficulties for general surgery or even for ear, nose, and throat surgery for which an oral endotracheal tube is inserted. For nitrous oxide given in the dental chair however a difficulty is encountered in that the patient cannot breathe through the nose when the mouth is propped open. Anæsthesia here should be induced with either thiopentone or ethyl chloride and then a nose-piece applied and nitrous oxide given. Although anæsthesia cannot be maintained indefinitely it can at least be prolonged if nitrous oxide is supplied under moderate pressure.

### STATUS LYMPHATICUS

This subject is discussed in Chapter XIII

### REFERENCE

- (1) Courville C B 1939 *Untoward Effects of Nitrous Oxide Anesthesia*  
Mountain View California



the bed and the bed pulled away from the wall so that the anæsthetist can stand behind and support the head. When the patient is recumbent there may be difficulty in maintaining a clear airway owing to the greater liability of the tongue to fall or be pushed backwards. There is an increased danger of the intrusion of a foreign body into the air passages. If anæsthesia is light blood or mucus gravitating backwards will irritate the posterior pharyngeal wall and make the patient restless.

## EPILEPSY

Epilepsy is not a contra indication to any form of general anæsthesia. We have seen only one attack in any way connected with anæsthesia and that was on recovery from nitrous oxide used for dental extractions. If a patient is known to be an epileptic it would be advisable to premedicate him with phenobarbitone or morphia.

## INSANITY

Induction of anæsthesia in an insane patient may sometimes be difficult if he is nervous or uncontrolled but once anæsthesia has been attained he is no more difficult than the normal subject. If a good vein is available we recommend that the patient's arm be steadied if necessary by force whilst anæsthesia is induced by thiopentone. If a patient is turbulent and it is necessary to use an inhalation anæsthetic for induction ethyl chloride owing to the rapidity of its action will be found the most humane.

Occasionally the administration of an anæsthetic is said to be responsible for the exacerbation of a psychosis or even for its onset. There is no evidence that an anæsthetic *per se* is ever the responsible factor but the mental stress associated with fear of anæsthesia and an operation in an unstable individual may precipitate a breakdown. Permanent cerebral degeneration has followed prolonged anæsthesia with nitrous oxide in which oxygenation has been inadequate.<sup>1</sup> These tragedies do not incriminate the anæsthetic itself but its injudicious use. Similar changes can be produced by simple oxygen deprivation if continued for a sufficient length of time.

## CLAUSTROPHOBIA

The anæsthetist occasionally has to deal with a claustrophobic patient who is terrified when a mask is placed over his face. This difficulty can be successfully overcome by inducing anæsthesia with

It is a mistake to restrict the use of basal anaesthesia in children solely to major surgery. Unfortunately there are general practitioners who ridicule the idea of taking the trouble of giving a basal anaesthetic for a trivial operation. They do not appreciate sufficiently that a child, particularly if he has had a previous unpleasant experience, is less concerned with the nature of the operation than with the terrifying prospect of being rendered unconscious. This applies equally to many adults.

For a particularly difficult child the advantages of basal anaesthesia, even for minor dental surgery, should be discussed with the parents. The mother should be made to realise that although an inhalation anaesthetic can safely be forced on an unwilling and resistant child, he may suffer psychological trauma, and that the responsibility for this would be hers. Psychological disturbance can be entirely avoided by the use of a basal anaesthetic and this should be advised if the parents are willing and able to bear the extra expense.

The spoilt child is often one whose teeth have been neglected. The dentist may not have had an opportunity of examining the mouth, or he may have been able to conduct only such a superficial examination that he could not decide which teeth could be saved and which were beyond saving. Any necessary dental treatment including examination, conservative work and extractions, can be finished in a single session under basal anaesthesia continued by endotracheal inhalation anaesthesia. Further the operation can be performed satisfactorily at the child's home.

The successful administration of a basal anaesthetic depends as much on the tact of the nurse as on the type of child. It is common for the mother to give her opinion that the child will object to a rectal injection more than to an inhalation anaesthetic, only to find later that it has been accomplished without protest.

For basal anaesthesia in children avertin and paraldehyde each has its supporters. We prefer the former possibly merely because we have had more experience with it. The technique of administration and dosage are discussed in the chapter on Premedication (p 102). Nembutal by mouth (p 104) is an alternative to avertin and paraldehyde by rectum but the certainty of success is not so great. However if all concerned co-operate well there is every prospect of a satisfactory result.

Ideally the basal anaesthetic should be given at the child's usual bedtime so that his day may be interfered with as little as possible. If the house is in a turmoil about the operation and if the child's usual routine is disturbed for example if he is put to bed early his

## CHAPTER XII

### CHILDREN

ALTHOUGH the drugs used to produce anæsthesia and the methods of their administration are the same for children as for adults there are different indications for their use. The problems of anæsthesia in dentistry are simplified in that extractions are usually easy and because children have not yet developed the habits and indulgences of later life which lead to resistance to anæsthesia. Nevertheless children present difficulties all their own.

Intelligent co-operation which plays a large part in successful anæsthesia particularly in the dental chair cannot be expected from the very young nor from the spoilt child. A harassed mother may communicate her fears to the child and make him a correspondingly more difficult patient. Since children under seven or eight years of age over react to a diminution of oxygen intake the administration of nitrous oxide to them is often exceedingly difficult. It is a well known clinical fact that if anæsthesia in the young unpremedicated child is established by nitrous oxide it is seldom possible to give sufficient oxygen to inhibit the jerky muscular movements resembling the jactitations of the deeply cyanosed adult without lightening anæsthesia so that the patient responds to surgical stimuli. These movements may involve the arms legs shoulders and back to such an extent that operating becomes almost impossible. Numerous explanations none of them entirely satisfactory have been given to account for the early onset of these asphyxial movements in childhood. Since the spinal reflexes are known to be augmented during asphyxia the instability of the nervous system in the very young is as good an explanation as any other.

If the child is being anæsthetised for the first time the credit or blame for the mental impression created must belong in great part to the anæsthetist. Basal anæsthesia provides such a pleasant transition to unconsciousness that it is specially indicated for children although at present not used as much as it should be. For a nervous child who has not been anæsthetised before it is ideal because it leaves only a pleasant memory of the onset of unconsciousness while the confidence of a child who has had a previous unfortunate experience of anæsthesia often can be regained by employing basal anæsthesia.

may disturb digestion and the child may come to the surgery with a full stomach because food taken hours earlier has not been digested. It is equally mistaken not to tell a child anything of what is to happen until he arrives in the dental surgery, since he may then fail in the future to have confidence in the dentist and anaesthetist. He should lead as normal a life as possible until an hour or so before the anaesthetic, and on his way to the surgery should be told that he is going to have some teeth out, but he will not feel anything of it because he will be enjoying a pleasant sleep.

On arrival the patient should always be given a chance to empty his bladder, since involuntary micturition is commoner in children than in adults.

It is generally advisable to persuade the mother or guardian to leave the surgery before the anaesthetic is started, since the child instinctively seeks maternal protection, and may cry and sob as long as the mother is present. Very often when she leaves the room, the previously unmanageable patient submits to treatment without any more ado. After the operation it is often helpful to allow the mother to come back and comfort the child while he is recovering from the anaesthetic.

The choice of an anaesthetic for dental surgery for a child is determined by the degree of co-operation which can be obtained by the difficulties of the extractions and by the parent's insistence on remaining in the room. If the parent remains in the room, not only is the child less likely to settle down but he may be penalised by the necessity for considering the mother's feelings for movement or moaning of the child during a light but adequate anaesthesia may make it difficult to convince her that he is not feeling pain, so that she may faint or interfere with the dentist during the extractions, or may afterwards tell the child she is sorry he was hurt. The deeper and more tranquil anaesthesia obtainable by the addition of ethyl chloride will avoid such complications. This can be done conveniently by giving  $N_2O$  nasally and at the same time covering the mouth with the upturned bib (fig 42 p 162) on which the ethyl chloride is sprayed. Even a small amount of ethyl chloride prevents or abolishes these movements or noises. The child may pay however for the parent's peace of mind by not feeling so well afterwards as if  $N_2O$  alone had been given. The presence of the parent may even lead to a decision to use ethyl chloride or vinesthene as the *sole* anaesthetic partly because the anaesthesia is tranquil and partly because cyanosis and jactitation will be avoided. The mother will be reassured by the unchanged colour of the child and by the resemblance of respirations to those in sleep.

Fortunately extractions in children are generally simple so that

gastric functions may be upset. The last meal of the day should be a light one taken two or three hours before the basal anæsthetic. The timing of the operation to correspond with the child's bedtime may give inconvenience to dentist and anæsthetist but it plays a considerable part in minimising the child's suspicions and so contributes to the success of the operation.

If the basal anæsthetic is to be administered rectally this is done about half an hour before the operation is due and the room is kept quiet and darkened until the child is unconscious. If nembutal by mouth is decided on it is mixed with honey and orange juice and should be given by the mother or nurse half to three quarters of an hour before the child's usual bedtime. He is then allowed to play about as usual under supervision. When he shows signs of drowsiness he can without difficulty be persuaded to lie down when someone may read to him. As soon as he is asleep he is undressed and put to bed. The inhalation anæsthetic should be commenced any time within the next half hour.

The dentist and anæsthetist with their apparatus remain out of sight in an adjoining room until the child is unconscious. After anæsthesia has been deepened by an inhalation anæsthetic an endotracheal tube is passed and anæsthesia maintained by nitrous oxide with the addition of ether if necessary. After the larynx has been packed off the dentist can make a thorough examination before proceeding with the necessary work. Even if conservative dentistry only is to be performed the larynx must be packed off to prevent tooth fragments or amalgam from being inhaled. A considerable time must be allowed for this type of operation for if the mouth is in bad condition the dentist may require two to three hours. He may drill a tooth for many minutes only to find that it is beyond repair and must be extracted. Teeth which have been drilled should be filled and the whole mouth put in order at the one sitting.

When the operation is over the nurse should remain with the child at least until he has recovered consciousness and it is best for her to remain throughout the night. Provided the child is not in pain the anæsthesia will merge smoothly into natural sleep and when he wakes on the following morning he will be unaware that anything unusual has happened and may be allowed to pursue his normal day's routine.

Unless basal anæsthesia is to be used the child should always be told beforehand that he is going to have an anæsthetic. The ambulatory child patient for dental surgery should be informed of this before he actually arrives in the surgery but not too long beforehand or he may worry about it even though he does not talk about it. Such fear

may disturb digestion and the child may come to the surgery with a full stomach because food taken hours earlier has not been digested. It is equally mistaken not to tell a child anything of what is to happen until he arrives in the dental surgery since he may then fail in the future to have confidence in the dentist and anæsthetist. He should lead as normal a life as possible until an hour or so before the anæsthetic, and on his way to the surgery should be told that he is going to have some teeth out but he will not feel anything of it because he will be enjoying a pleasant sleep.

On arrival the patient should always be given a chance to empty his bladder since involuntary micturition is commoner in children than in adults.

It is generally advisable to persuade the mother or guardian to leave the surgery before the anæsthetic is started since the child instinctively seeks maternal protection and may cry and sob as long as the mother is present. Very often when she leaves the room, the previously unmanageable patient submits to treatment without any more ado. After the operation it is often helpful to allow the mother to come back and comfort the child while he is recovering from the anæsthetic.

The choice of an anæsthetic for dental surgery for a child is determined by the degree of co-operation which can be obtained, by the difficulties of the extractions and by the parent's insistence on remaining in the room. If the parent remains in the room not only is the child less likely to settle down but he may be penalised by the necessity for considering the mother's feelings for movement or moaning of the child during a light but adequate anæsthesia may make it difficult to convince her that he is not feeling pain so that she may faint or interfere with the dentist during the extractions or may afterwards tell the child she is sorry he was hurt. The deeper and more tranquil anæsthesia obtainable by the addition of ethyl chloride will avoid such complications. This can be done conveniently by giving  $N_2O$  nasally and at the same time covering the mouth with the upturned bib (fig 42 p 162) on which the ethyl chloride is sprayed. Even a small amount of ethyl chloride prevents or abolishes these movements or noises. The child may pay however for the parent's peace of mind by not feeling so well afterwards as if  $N_2O$  alone had been given. The presence of the parent may even lead to a decision to use ethyl chloride or vinesthene as the *sole* anæsthetic partly because the anæsthesia is tranquil and partly because cyanosis and jactitation will be avoided. The mother will be reassured by the unchanged colour of the child and by the resemblance of respirations to those in sleep.

Fortunately extractions in children are generally simple so that

N<sub>2</sub>O is often adequate. If however perfectly tranquil anæsthesia for a few minutes is demanded nitrous oxide should not be chosen for a very young or obstructive patient. It is impossible to persuade such children to breathe through the nose, and the use of a mouth piece to give the gas through the mouth is unsatisfactory because its necessarily tight application is frightening and often difficult when the child is throwing his head from side to side. In such cases ethyl chloride or vinesthene should be used. They have the advantages that either can be administered with ease even when the patient is obstreperous, that anæsthesia is quickly established and that the operating conditions afforded are entirely satisfactory. We have sometimes used intravenous anæsthesia in children as young as 5 but its use in the young is limited because there is often not a large enough vein into which the solution can be injected and because the child often refuses to keep his arm still.

When there is only one tooth to be extracted and the case is simple, a few seconds unconsciousness suffices. In such cases it is immaterial whether nitrous oxide, ethyl chloride or vinesthene is chosen since with the very small amount necessary the patient wakes immediately without unpleasant after effects.

It is important to remember that the prospect of repeated anæsthesia commends itself even less to a child than to an adult and that it is generally advisable to finish all necessary extractions at one sitting if possible. Although a child may be coaxed into facing one or possibly two sittings he cannot reasonably be expected to co operate in three or four.

A child should not be hurried and if he is co operative everything should be explained to him and nothing forced on him unexpectedly. Even a few minutes unhurried chat with an averagely intelligent child will usually secure his co operation and make it possible to administer nitrous oxide nasally.

For a very small child the head rest should be taken off and the nape of his neck rested on a cushion on the back of the chair. The child can be raised the required amount by seating him on telephone directories or cushions placed on the chair (fig 40 p 160).

Every anæsthetist has his own tricks for inducing an effective anæsthesia pleasantly. For example if nitrous oxide is to be given to a boy he can be shown the rebreathing bag and told to blow it up like a football in as few breaths as possible but entirely through his nose and that we would like him to beat the previous record of ten breaths. A prize may be offered as an incentive. As he expires through his nose he is encouraged by the administrator counting his expirations.

aloud. If the patient expires through the mouth, the anæsthetist at once says: "We can't count that, you know."

Children who have been dreading the anæsthesia and yet have been plucky enough to conceal their fears are often difficult, particularly under  $N_2O$ , just as consciousness is being lost. Then there may be restlessness until anæsthesia is fully established and an apprehensive child may even vomit food eaten several hours before. Warnings that vomiting may occur are slight irregularity of breathing, pallor, and swallowing or retching. If vomiting occurs before extractions are commenced it should be allowed to finish and the anæsthetic and operation can then be continued as described on p. 233.

If the child is unwilling or unable to co-operate and if it is desired to proceed at once with the operation, the easiest method of anæsthetising him is to spray ethyl chloride over an open mask or over the up-turned bib adjusted over his nose and mouth (fig. 43 p. 163).

If it is thought undesirable to place the patient in the dental chair for the induction of anæsthesia, the dentist (or nurse) can sit on a settee or the dental chair and take the child on his lap. His sole function is to hold the patient's wrists so that the hands cannot pull the mask away from his face. The child may kick and damage the shins of the person on whose lap he is sitting but this can be easily avoided if the holder encircles the patient's legs with his own. The patient held in this way can move only his head which can be followed easily by the anæsthetist and the patient soon rendered unconscious. When this stage is reached he is carried and placed in the usual position in the dental chair.



FIG. 27



## CHAPTER XIII

### STATUS LYMPHATICUS

STATUS lymphaticus was first described in modern medicine in 1889 by Paltauf<sup>1</sup> as a syndrome characterised by an enlarged thymus gland generalised lymphoid hyperplasia vascular hypoplasia and a susceptibility to sudden death from trivial causes. In fatalities which have been attributed to this syndrome almost invariably the patient has been a child which at first sight one might be tempted to call a fine child but qualifies the praise adding rather pasty perhaps.<sup>2</sup> During an operation however slight this may be e.g. for removal of tonsils and adenoids or even late in induction he is noticed to be pale he soon stops breathing and unless resuscitative measures are instituted immediately he dies. At the subsequent investigation the anaesthetist states that he has given the anaesthetic in his usual manner. The autopsy reveals no abnormality beyond hyperplasia of the thymus gland and lymphoid tissue generally. These findings are considered by some pathologists and coroners to provide a sufficient explanation for death under anaesthesia and as exonerating the anaesthetist from any responsibility for the death.

In 1926 a committee was set up by the Medical Research Council and the Pathological Society of Great Britain and Ireland to enquire whether any such condition as status lymphaticus really exists. Examination of the available statistics taking into account the normal limits of the weight of the thymus at different ages showed that in the twenty three cases analysed of death during anaesthesia or as the result of shock the thymus could be considered abnormally large in only four and even in these four there was no satisfactory proof that the large thymus played a part in accounting for the deaths. No correlation was found between the weight of the thymus and the amount of lymphatic tissue elsewhere in the body. The Committee concluded<sup>3</sup> that there was no evidence that status lymphaticus exists as a pathological entity.

The views expressed in the previous two paragraphs are irreconcilable. We should not be justified in expressing an opinion on pathological findings but we think it proper to make certain observations on the clinical reactions of children to anaesthesia based on our

experience of many thousand anæsthetics given to children for dental and for tonsil and adenoid operations

It is unprofitable to attempt to prove a negative but if by the term status lymphaticus is implied a condition in which the administration of an anæsthetic *inevitably* causes death—an impression so frequently conveyed by the coroner's verdict—then in our opinion no such state exists. Nevertheless some children die as a result of slight traumatic or psychological insults which would have little or no effect on others. This category would include the rare instances in which death is alleged to be due to operative shock under light anæsthesia. We ourselves have no first-hand knowledge of such cases but as anæsthetists we recognise a *status periculosus* in which the child shows a susceptibility to the depressant effects of anæsthetics which amounts to an idiosyncrasy. With this child a little anæsthetic goes a long way—what would be a safe amount for a normal child for him may prove rapidly fatal. We have seen about twelve cases of collapse early in anæsthesia in such children where the prompt institution of treatment brought about recovery and permitted the completion of the operation. We feel however that if death had occurred in any of these cases status lymphaticus might well have been diagnosed and that the anæsthetist would have been exonerated from any blame. Although these twelve cases had certain characteristics in common we are not able to describe them as a clinical type in which abnormal reaction to anæsthesia can reasonably be anticipated because a very large number of children with apparently similar characteristics do not show this idiosyncrasy. Our outstanding impression of the twelve children is that they exhibited an unmoderate docility. These children who show no fight must be anæsthetised with extreme care. The reactions which commonly occur during the induction period are not seen. Instead of flushing pallor is remarked, respiration is shallow and regular from the beginning, resistance is strikingly absent and there is an almost imperceptible merging into unconsciousness which supervenes after an unusually small amount of anæsthetic has been given. This sudden and unpredictable response to a small dose of anæsthetic may easily be overlooked and a small additional amount of anæsthetic results in extreme loss of muscular tone accompanied by wide dilatation of the pupils. We believe such a patient can be anæsthetised with safety provided the amount of anæsthetic administered is carefully limited to the quantity required for the satisfactory performance of the operation and we consider that when death occurs it is due to the effect of an overdose of anæsthetic upon a susceptible patient.

Since this undue sensitivity to anæsthetic agents produces a liability to overdosage it is imperative that the anæsthetist understand what measures are to be taken should respiration fail. These are the same as in any other case of respiratory arrest under anæsthesia namely provision of an absolutely clear airway, the institution of artificial respiration and administration of oxygen. This treatment applied promptly will save life in those cases in which a large overdose of anæsthetic has not been given.

It is our opinion that in deaths under anæsthesia the anæsthetist is more responsible than either the drug used or the pathological condition of the patient (always excepting the unusual emergency where the patient is pulseless and practically moribund before anæsthesia is begun). The following facts support the validity of this point of view even when applied to cases where an idiosyncrasy to anæsthetics admittedly exists. (i) Verdicts of 'status lymphaticus' have been brought in as an apparently satisfactory explanation of death under anæsthesia in children who have previously withstood successfully the effects of an operation under general anæsthesia. (ii) Often when a child has collapsed under anæsthesia the operation has been abandoned solely through fear that he was *in statu lymphatico* and therefore that death probably would occur if the anæsthetic were continued. In some of these cases operation became imperative and a second anæsthetic given a few days later has been entirely successful. (iii) We have not heard of an anæsthetist in whose practice has occurred more than one death attributed to this cause. Such unfortunate coincidences no doubt have been recorded but the general failure of individual practitioners to encounter repetitions of this catastrophe appears to indicate that the experience gained in dealing with one case confers upon the anæsthetist the ability to avoid recurrences. But the admission that death from status lymphaticus is avoidable destroys the very argument upon which belief in the existence of such a condition rests. (iv) Routine autopsy upon the victims of accidents where the extent of the injuries (e.g. ruptured liver) would have made death inevitable in the most robust individual has occasionally disclosed a degree of lymphoid hyperplasia greater than that upon which a verdict of status lymphaticus has been founded in cases of death under anæsthesia. Nevertheless on enquiry many of these individuals in whom lymphoid hyperplasia is revealed by chance are found at some time or other to have negotiated successfully the hazards of anæsthesia and operation without exhibiting an undue tendency to succumb to either.

In a large percentage of all cases of death under anæsthetics

autopsy fails to reveal any cause for death. In death from overdose of an anæsthetic for example there is no diagnostic autopsy finding. The coroner finds his task simplified if the pathologist can state that the autopsy revealed a definite condition which could account for the death, and hyperplasia of the lymphatic system is at present accepted by many as one such condition. Partly because of his desire to report some positive finding partly because of his sympathy for the anæsthetist and partly because of public policy, a pathologist may diagnose status lymphaticus when no convincing explanation for death is found. This diagnosis which in some quarters has now come to carry with it the suggestion that death was inevitable and that no one was to blame may serve a useful purpose for the reasons indicated but it must not be overlooked that in these cases a detailed anæsthetic history of the sequence of events prior to death is far more illuminating than the most searching autopsy.

According to Greenwood\* the deaths now ascribed to status lymphaticus would in more pious but not more superstitious days have been attributed to the visitation of God ' a verdict which he suggests would be ' at least as scientific as and more modest than status lymphaticus ' . To this opinion we add our suggestion that deaths under anæsthesia attributed to status lymphaticus may well be due to nothing more nor less than overdosage in a susceptible patient.

## REFERENCES

- (1) Paltauf A 1889 *Wien klin Wschr* 2, 877 1890 *Ibid* 3 172
- (2) Greenwood M and Woods H M 1927 *J Hyg Camb* 26, 305
- (3) Young M and Turnbull H M 1931 *J Path Bact* 34 213

Read also

- Cohen H 1936 *Practitioner* 136 252  
 Cohen H 1939 *British Encyclopædia of Medical Practice* London  
 12 23

Since this undue sensitivity to anæsthetic agents produces a liability to overdosage it is imperative that the anæsthetist understand what measures are to be taken should respiration fail. These are the same as in any other case of respiratory arrest under anæsthesia namely, provision of an absolutely clear airway the institution of artificial respiration, and administration of oxygen. This treatment applied promptly, will save life in those cases in which a large overdose of anæsthetic has not been given.

It is our opinion that in deaths under anæsthesia the anæsthetist is more responsible than either the drug used or the pathological condition of the patient (always excepting the unusual emergency where the patient is pulseless and practically moribund before anæsthesia is begun). The following facts support the validity of this point of view even when applied to cases where an idiosyncrasy to anæsthetics admittedly exists. (i) Verdicts of *status lymphaticus* have been brought in as an apparently satisfactory explanation of death under anæsthesia in children who have previously withstood successfully the effects of an operation under general anæsthesia. (ii) Often when a child has collapsed under anæsthesia the operation has been abandoned solely through fear that he was *in statu lymphatico* and therefore that death probably would occur if the anæsthetic were continued. In some of these cases operation became imperative and a second anæsthetic given a few days later has been entirely successful. (iii) We have not heard of an anæsthetist in whose practice has occurred more than one death attributed to this cause. Such unfortunate coincidences no doubt have been recorded but the general failure of individual practitioners to encounter repetitions of this catastrophe appears to indicate that the experience gained in dealing with one case confers upon the anæsthetist the ability to avoid recurrences. But the admission that death from *status lymphaticus* is avoidable destroys the very argument upon which belief in the existence of such a condition rests. (iv) Routine autopsy upon the victims of accidents where the extent of the injuries (e.g. ruptured liver) would have made death inevitable in the most robust individual has occasionally disclosed a degree of lymphoid hyperplasia greater than that upon which a verdict of *status lymphaticus* has been founded in cases of death under anæsthesia. Nevertheless on enquiry many of these individuals in whom lymphoid hyperplasia is revealed by chance are found at some time or other to have negotiated successfully the hazards of anæsthesia and operation without exhibiting an undue tendency to succumb to either.

In a large percentage of all cases of death under anæsthetics

Many dentists and anæsthetists consider that the taking of certain articles of diet predisposes to post operative vomiting. Even milk, beef tea or some equally innocuous food may be prohibited because post-operative vomiting has occurred on some previous occasion although adequate enquiry in all probability would have revealed that the patient had been abnormally apprehensive. Instead of banning any such item, it is more rational to arrange for pre-operative treatment calculated to allay the nervousness which is responsible for a derangement of normal digestive function and a tendency to post-operative vomiting.

Such preparation is especially necessary for a patient with a history of having vomited previously after nitrous oxide anæsthesia. If on a second occasion special care is taken to allay apprehension it is rare for vomiting to occur.

One of us was called in to anæsthetise a nervous woman who gave a history of vomiting after gas. The patient was first seen in the dental surgery and there was therefore no opportunity to premedicate her. Anæsthesia with nasal nitrous oxide was short and two teeth were extracted. After recovering consciousness the patient vomited a small meal taken six hours previously. To prepare her for a further tooth extraction the following week she was given a prescription for a mixture containing chloral hydrate 5 gr. and potassium bromide 10 gr. to the ounce of peppermint water. She was instructed that on the two days preceding the next anæsthetic she should take one ounce of the mixture three times a day after meals. With this premedication the next session passed off uneventfully.

## THE HOSPITALISED PATIENT

It is often difficult particularly for a dental operation to persuade the patient to enter the hospital or nursing home even for one night beforehand and frequently he is admitted about lunch time for operation in the late afternoon. Experience shows that the longer the patient spends in the institution prior to operation the smoother is the period of anæsthesia and the more rapid the convalescence. Apart from the considerable advantage conferred by the rest the patient benefits from being given correct diet and appropriate sedative drugs.

Before minor surgery or major dental surgery, if the patient is not constipated neither a purgative nor an enema is necessary but if constipated he should be given an enema in the early morning on the day of operation. No special diet is necessary beyond the regime

## CHAPTER XIV

### PREPARATION FOR OPERATION

BEFORE a minor operation it is desirable and before a major operation essential for a patient to receive treatment which will reduce his apprehension and appreciation of the preliminaries of anæsthesia and make him a suitable subject both for anæsthesia and for operation. The degree of preparation depends on the nature and extent of the operation to which the patient is to be subjected and on the anæsthetic chosen. Premedication an important feature of the preparation of a patient is dealt with elsewhere. Although for the ambulatory patient little or no preparation may be actually necessary it will make anæsthesia more tranquil and at the same time make recovery less unpleasant. For the hospitalised patient pre operative preparation is a matter of routine.

#### THE AMBULATORY PATIENT

For nitrous oxide anæsthesia and a tooth extraction of average difficulty the average patient requires no physical preparation at all. The more abnormally resistant the patient and the longer the period for which anæsthesia is expected to be necessary the more important does it become to prepare him for the occasion. The sedative drugs suitable for the ambulatory patient are discussed on p 93. Purgation is neither necessary nor advisable. Unfortunately the old fashioned prejudice in favour of starvation before anæsthesia still persists and the patient may in consequence come to the dental chair weak from lack of food. In the ambulatory patient starvation is in fact a much more frequent cause of post operative complications such as nausea retching faintness or collapse than is the presence of food in the stomach at the time of operation. The patient therefore should take an average meal three or four hours before operation and if apprehension has been prevented by appropriate premedication digestive activity will continue undisturbed and the stomach will be empty by the time the operation is due. If this period of abstinence from food must be exceeded some sugar containing food should be given two hours before operation for example a cup of tea containing three or four lumps of sugar. Alternatively an ounce of barley sugar or half a glass of orange juice may be taken.

## CHAPTER XV

### BARBITURATES

BARBITURIC acid compounds first synthesised in 1902 have continued to increase in popularity as hypnotics. Barbitone better known as Veronal was the first with which success was achieved. Its chemical formula is



the essential malonyl urea radicle of barbituric acid being represented as B. By the substitution of other groups for one or both of the  $\text{C}_2\text{H}_5$  (ethyl) groups of veronal new barbiturate hypnotics have been produced. For example phenyl ethyl barbiturate or phenobarbitone has the formula



The barbiturates are sedatives but since the potency sites of action, and rates of absorption and excretion of different members of this group of compounds vary considerably there are correspondingly marked differences in their pharmacological actions. For example, veronal because it is only slightly soluble in water is therefore slowly absorbed and has a correspondingly delayed action. On the other hand medinal a salt of veronal and much more soluble in water than the latter is more rapidly absorbed and because of its quicker action has become more popular than veronal. Both these drugs are typical of the long acting barbiturates so called because of their slow excretion. The number of barbiturate compounds now in use is great and discussion of them is complicated by the confusion which has arisen because different proprietary names have been given to drugs the actions of which are clinically indistinguishable. In fact in some cases the same barbiturate manufactured by different commercial houses is known by different names for instance phenobarbitone can be purchased either as Luminal or as Gardenal.

Greater structural alteration than had previously been made in



to which a poor sailor would subject himself before facing a rough sea crossing. The patient may have a light supper the night before operation and if this is timed to take place in the morning about three hours previously he can take for breakfast a cup of tea well sugared and a piece of bread and butter. If the operation is timed for the early afternoon he can have an ordinary light breakfast and a cup of tea or soup in the middle of the morning. A sedative (e.g. phenobarbitone 1 gr) should be given on the evening before operation. Pre-operative medication on the day of operation is discussed on p. 95 et seq.

For a **major abdominal operation** the patient should be in the nursing home for the two previous nights. Extensive pre-operative purgation and starvation are now things of the past. The modern custom is to give a mild aperient preferably one to which the patient is accustomed on the evening of the day but one before operation and a soap and water enema early on the morning of the day of operation.

A sedative should be given on each of the two evenings before the operation on the first evening aspirin 10 gr or phenobarbitone 1 gr and on the next a more powerful one (medinal 10 gr or nembutal 3 gr).

On the day before operation the patient should have chicken or fish for the main meal in the middle of the day and a light evening meal. If the operation is to be in the morning a cup of tea can be given at 7 a.m. and if in the afternoon the patient may have another drink in the middle of the morning.

dissolves immediately. The strength of solution most commonly recommended is 5 per cent, but we have used solutions of strengths between 2½ and 20 per cent and have observed no differences in the character of the resulting anæsthesia nor in the incidence of post-operative complications. When the dose of thiopentone is to be given quickly, it is immaterial what strength of solution is used, but if anæsthesia is to be continued for some time by intravenous injection the use of dilute solutions makes it easier to control the dosage and so to maintain a uniform level of anæsthesia.

The extreme ease with which the ultra short-acting barbiturates can be administered constitutes one of the dangers attendant upon their use and should never tempt the operator to act as his own anæsthetist. The safeguarding of an unconscious patient is a whole-time responsibility. When these barbiturates were first introduced the opinion was generally held that the simplicity of the technique of their administration would reduce the scope of the specialist anæsthetist. Experience has shown however that the use of these drugs should be confined to those who have a sound knowledge of anæsthetic drugs and anæsthetic routine generally.

Thiopentone may be given to a patient of any age but this is rarely practicable in children under six both because their co-operation is difficult to obtain and also because their veins are seldom large enough to enable an intravenous injection to be given with certainty. Although the production of the state of anæsthesia with thiopentone is invariably easy if it is possible to locate a vein into which an injection can be given, the patient should not be *promised* this drug before it has been ascertained that his veins are suitable.

Thiopentone given intravenously may be used

**1 As a basal anæsthetic before a major operation**—The dose to be given depends on whether anæsthesia is to be continued at once with an inhalation agent such as ether or whether the period of basal anæsthesia has to be prolonged to allow the transport of the unconscious patient from his bed to the operating theatre. In the first case 0.3–0.4 g. will usually be found sufficient and in the second case the dose must be increased but should rarely exceed 0.7 g.

**2 As the sole anæsthetic for a prolonged operation**—This method of anæsthesia has been prevented from becoming popular by the difficulty of simultaneously superintending a continuous intravenous injection and maintaining a clear airway and because the rate of recovery of consciousness after large doses of thiopentone is slow.

the substitution groups resulted in the production in 1928 of Sodium Amytal and in 1930 of Nembutal in America and of Pernocton in Germany. These are very soluble in water, and their administration intravenously leads within a few minutes to unconsciousness. Each of these short acting barbiturates soon became popular as a basal anæsthetic but low therapeutic quotients (p 17) render them unsuitable as sole anæsthetics for the production of general anæsthesia. Further they are slowly excreted so that even after their use as basal anæsthetics return to consciousness is delayed for two to three hours longer than if they had not been given. Even for production of basal anæsthesia these drugs have now been largely superseded by the ultra-short acting barbiturates.

Evipan (Hexobarbitone B P) the first of the ultra short acting barbiturates introduced in Germany in 1932 was followed by Pentothal (Thiopentone B P) a thio barbiturate (sulphur containing barbiturate) from America in 1935. These drugs are extremely soluble both in water and in lipoids and the therapeutic quotient of each is high. They can be given by mouth but because there are marked variations in the rate of absorption from the alimentary tract in different individuals and in different circumstances the intravenous route is safer, more accurate and more reliable. Drowsiness, sleep or anæsthesia of varying degree results according to the dose injected.

Thiopentone is a more satisfactory drug than hexobarbitone in that its use is not accompanied by the slight muscular twitches occasionally seen during hexobarbitone anæsthesia. Moreover thiopentone produces greater muscular relaxation and more peaceful anæsthesia. However it causes a greater depression of the respiratory centre than does hexobarbitone and if a solution of thiopentone is in error deposited extraveneously the local tissue reaction is more severe than after the same accident with hexobarbitone. Except for these differences the actions of these drugs are so similar (although thiopentone is rather more potent than hexobarbitone) that we shall refer only to thiopentone but observations on it can be taken as applying equally to both.

### SOLUBLE THIOPENTONE

Soluble thiopentone B P is now made by several firms. The original manufacturers gave the name Pentothal to their preparation. This drug is supplied as a sterile powder in ampoules containing 0.5 or 1 g. Ampoules containing 10 c.c. or 20 c.c. of sterile distilled water are supplied separately. When water is added to it the powder

to  $N_2O$  for a short operation such as a dental extraction—In describing the technique for the administration of thiopentone we shall deal with this latter use of it which has a special application to the ambulatory patient in the dental surgery. There is here the additional problem of enabling the patient to return home within a short time after the operation is completed.

When a quick recovery is desired it is our practice to inject rapidly the whole of the dose which we estimate will be needed just to produce unconsciousness and immediately the latter is attained to apply the nose piece and to continue anaesthesia with nitrous oxide. The dose varies from 0.1 g. in the frail to 0.5 g. in the robust alcoholic patient. It is important to recognise that the anaesthetic effect produced by this dosage may be slight and in any case it will be only transient. The amount given should be such that once it is injected anaesthesia can be maintained only by continuing at once with  $N_2O$  given in the usual way as though it had not been preceded by thiopentone.

The opinion that thiopentone has no place in the dental surgery because of the slow recovery and because of the lowering of blood-pressure which large doses may induce (particularly in patients in the upright position) is not now tenable. Each of these disadvantages is avoided by the use of thiopentone in small dosage given rapidly prior to  $N_2O$  anaesthesia. A pleasant induction to anaesthesia is assured, recovery is rapid and when the patient awakes he is unaware of any feature of anaesthesia beyond the beginning of the injection.

The use of a preliminary dose of thiopentone is particularly valuable for the anaesthetic-resistant patient. In this case sufficient should be given actually to assist the nitrous-oxide anaesthesia. On an average 0.3 to 0.5 g. of either thiopentone or hexobarbitone is required and should be given moderately rapidly. The nose piece is then put on and nitrous oxide administered. After a dose of 0.3 g. given quickly to a powerful man nitrous oxide anaesthesia can be continued in the usual way and the effect of this small amount of barbiturate can be disregarded but when a larger dose is given oxygen deprivation should not be allowed. On recovery the patient should rest until the effect of the anaesthetic has worn off usually about twenty minutes to half an hour.

Although recovery from thiopentone is almost invariably peaceful the anaesthetic resistant subject may during recovery go through a brief period of drunken aggressiveness during which he has to be forcibly restrained.

The occurrence of vomiting after average doses of thiopentone is rare. We have however encountered a few patients in whom the

The total dosage of thiopentone given for an operation for appendicectomy which lasted thirty five minutes was 2.4 g. Two anaesthetists were present one to give the intravenous injection over a period of half an hour the other to superintend the airway. In order to obtain muscular relaxation the dosage given was sufficient to produce noticeable respiratory depression at certain stages of the operation. The slow rate of recovery made it necessary for a nurse to be in constant attendance on the patient for five hours after completion of the operation.

**3 As the sole anaesthetic for a short operation** — The anaesthetic technique here is usually easy generally the dose estimated to be necessary is given by the anaesthetist in a single injection and immediately thereafter he devotes his attention to the maintenance of the airway.

Anæsthesia produced in this way is a contrast to that produced by inhalation agents in that the depth of anæsthesia does not remain at a steady level. It is at its maximum almost immediately after the injection and then becomes progressively lighter as the drug is eliminated.

Another disadvantage of this method is the necessity for calculating beforehand how much anaesthetic will be required for a given operation. For this the probable duration of the operation must be estimated and the reaction of the patient to the drug must be anticipated. If an incorrect estimate is made it may lead to the administration of too small a dose in which case the patient will begin to recover consciousness before the operation is completed or of an unnecessarily large dose providing adequate anaesthesia for the operation but inconveniently delaying recovery. For instance difficulties which will be encountered in a particular extraction may be greatly over estimated.

A colleague asked to be anaesthetised with hexobarbitone alone for extraction of a tooth and was given 8 c.c. of a 10 per cent solution (0.8 g.) it being estimated that the extraction would take seven or eight minutes. The tooth however was out in a minute but it was two hours before the patient had recovered sufficiently to leave the surgery. If an accurate estimate had been made of the length of anaesthesia needed only half the dose administered would have been given and the patient would have recovered completely in 20–30 minutes.

**4 As a very transient basal anaesthetic preparatory to a potent anaesthetic such as ether for a prolonged operation or**

above. A colleague in poor health had to undergo the extraction of a number of teeth at four sessions at weekly intervals. He refused premedication and insisted that anæsthesia should be with nitrous oxide alone. Although anæsthesia was uneventful on the first occasion, he disliked the prospect of its repetition. On the second occasion, in order to abolish his heightened reflexes a greater degree of anoxæmia had to be induced than was previously necessary and there was some post-anæsthetic collapse. The patient's dread of the necessity for reducing the oxygen intake during anæsthesia and post-operative prostration were still more marked on the third and fourth occasions.

It should be emphasised that in dental anæsthesia for the ambulatory patient thiopentone is indicated where *anæsthetic* difficulties are anticipated and not for cases in which the difficulty is that of extraction.

### Thiopentone in War Surgery

Thiopentone has been of the greatest value in war surgery, nevertheless the very ease with which it can be given has led to unnecessary deaths in the hands of those unfamiliar with the pitfalls of anæsthesia. The outstanding virtues of this drug are pleasantness of induction and the relative absence of post-anæsthetic vomiting. These have indeed proved a boon to patients in whom a series of operations, as for example for plastic surgery, has had to be performed. Other attractive qualities of the drug are its speed of action, portability and freedom from fire and explosion hazards. There are few patients who are not the better for being induced with thiopentone, but the decision to maintain anæsthesia with the same drug should not be taken lightly, particularly if deep or prolonged anæsthesia is required. With deep anæsthesia from this drug alone some degree of respiratory depression is inevitable. With an experienced anaesthetist this is of little consequence, but if the situation is handled unskilfully death can result. Prolonged anæsthesia from thiopentone alone is followed by a long interval of unconsciousness. This period is one of potential danger to the patient, particularly in the absence of skilled nursing.

### Contra-indications to the Use of Thiopentone

The short acting and ultra short-acting barbiturates are excreted or detoxified mainly by the liver and large doses should therefore be avoided in cases of liver deficiency. Although it is highly improbable that the first two conditions described below as contra-indications will

injection of a barbiturate intravenously has been followed by giddiness and bouts of vomiting lasting for as long as twenty-four hours

### Indications for the Use of Thiopentone in the Dental Surgery

In the great majority of patients for dental extractions anæsthetic difficulties are not encountered but it is not sufficiently appreciated outside the dental profession that there is a small percentage of cases where the problem of anæsthesia is by no means simple. Difficulties which can be overcome by the use of thiopentone as a transient basal anæsthetic arise in two totally different groups of patients. The first is the anæsthetic resistant group (p 113) in members of which even the experienced anæsthetist does not find it easy to provide five minutes tranquil anæsthesia with N<sub>2</sub>O alone. With this gas conditions adequate for operation can often be achieved only by pushing the anæsthetic until a degree of anoxæmia is produced which would be dangerous if it were maintained for any length of time or if it were increased. A small dose of thiopentone given intravenously before N<sub>2</sub>O anæsthesia is begun produces unconsciousness so rapidly that the stage of potential delirium is passed through quickly enough to eliminate struggling during induction. In addition the thiopentone enables tranquil anæsthesia thereafter to be maintained with nitrous oxide.

Patients of the second group have a horror of *inhalation* anæsthesia. Anæsthesia here presents no technical difficulties and can in fact be achieved and maintained with N<sub>2</sub>O with ease but the patients suffer mental distress both before and after administration. This group includes also patients who have to face a series of anæsthetics and who instead of deriving reassurance from experience dread each approaching inhalation anæsthetic more than the last even when the administration has been in skilful hands. This reaction described by older writers as cumulative resistance to anæsthetics is psychological and can be almost entirely abolished by the use of intravenous barbiturates.

It was necessary for a woman to have all her remaining teeth extracted and the work was planned to occupy five sittings over a period of a month. She was very apprehensive before the first occasion but since thiopentone was given prior to nitrous oxide anæsthesia the experience completely reassured her and she approached with equanimity the subsequent sessions at which the same technique was repeated.

The following case in which thiopentone was indicated but was refused by the patient provides a contrast to the case described

might well produce respiratory arrest in the same individual suffering from or recovering from shock.

If the above contra indications are observed there is no need before inducing anaesthesia with thiopentone to conduct any special physical examination of the patient, or to perform any particular tests beyond those to which he might be subjected before any other general anaesthetic.

### Technique of Administration of Thiopentone

If the operation is for dental extractions a prop should always be placed in the mouth immediately before anaesthesia is begun.



Fig 28

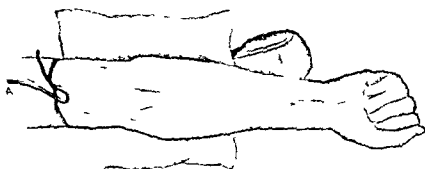


Fig 29

An assistant supports the patient's extended right elbow with his own right hand and with his left hand compresses the patient's right arm above the elbow tightly enough to prevent the return of venous blood (fig 28) but not so tightly that the flow of arterial blood into the arm is impeded. The patient should clench his fist to assist in making the veins as prominent as possible.



be encountered in dental practice it is essential to include them at the head of the list since the use of barbiturate anæsthesia in these cases may turn the scale against the patient

**1 Acute intestinal obstruction**—The liver function is disorganised by the action of toxins absorbed from the obstructed bowel

**2 Jaundice**—This condition is usually associated with gross disturbance of the liver function and should be regarded as a definite contra indication to the use of thiopentone We have however used the drug in very small doses without untoward effects but its excretion is extremely slow A dose of 0.15–0.20 g in an adult may produce unconsciousness lasting for several minutes

**3 As the sole anæsthetic for operations on the throat,** and for operations in which the protective laryngeal reflexes may be elicited as for example if a laryngoscope is to be used In our experience the motor response to such stimuli is hyper-active during light barbiturate anæsthesia and may lead to persistent and alarming laryngeal spasm

**4 Asthma**—There is experimental evidence that a general constriction of the bronchial musculature may result from the use of any barbiturate<sup>2,3</sup> For this reason many regard these drugs as contra indicated in asthma (p. 120)

**5 Anæmia, and marked debility**—These are only relative contra indications Anæsthesia in such patients is produced with only a small amount of any anæsthetic Doses of the barbiturates which would be correct for the average adult may be dangerously excessive for them and in any case will lead to post-operative depression and delayed recovery The dose given should therefore be reduced to correspond to the patient's lowered resistance to anæsthetics

Thiopentone must be given very cautiously to patients who have a sluggish peripheral circulation Here the drug takes much longer to reach the heart pass through the pulmonary circulation and return to the heart again whence it is distributed to the brain This increased latent period must not be incorrectly interpreted as tolerance on the part of the patient to the effects of the drug In fact the reverse is the case A patient with a failing circulation is susceptible to the effect of thiopentone which however takes longer in manifesting itself for purely mechanical reasons Owing to liver dysfunction consequent upon the failing heart thiopentone is excreted slowly and its action is therefore prolonged

In shock too even where resuscitation has been adequate and recovery has apparently taken place thiopentone must be used with discretion A dose which is appropriate for a robust subject

When the vein is entered in this way the skin puncture does not lie directly over the vein puncture, and the occurrence of bleeding after the needle is withdrawn is thus prevented

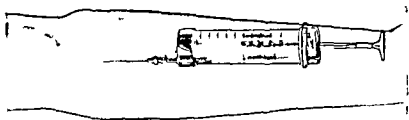


FIG 33

After a little practice, it is easy to recognise by sense of touch when the vein has been penetrated but in any case this should always be confirmed by withdrawing the plunger gently when if the needle is in the vein blood will be aspirated into the syringe

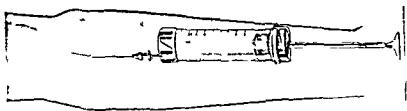


FIG 34

The syringe should not be filled completely with thiopentone solution, because then it will be impossible to withdraw the plunger to verify that the vein has been entered. The position of the plunger here should be contrasted with that shown in fig 33

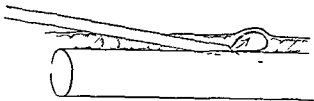


FIG 35

If the bevel of the needle does not lie completely within the lumen of the vein blood can be aspirated into the syringe but when the solution is injected some will be deposited extravascularly. If this

Alternatively the extended arm is supported by a pillow which rests on the arm of the dental chair. To distend the veins a piece of soft rubber tubing is tied round the upper arm (fig 29) in such a fashion that it can be released simply by a pull on one end of it (A). The skin is cleansed with spirit

FIGS 30 AND 31

Air must be expelled from the syringe before the needle is inserted into the vein

FIG 32

The syringe has an eccentric nozzle to allow the barrel to rest closely on the forearm so that if the patient moves his arm the relation of the needle to the vein will not be disturbed. The needle is almost parallel to the vein and before

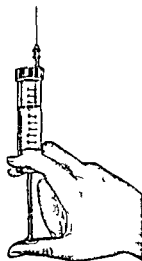


FIG 30

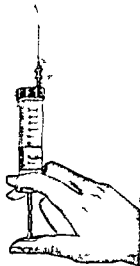


FIG 31

the skin puncture is made the vein is steadied in position by stretching the skin which lies over it. The needle is inserted through the skin a little to one side of the vein and it is then advanced alongside the vein for about  $\frac{1}{8}$  inch before being introduced into it

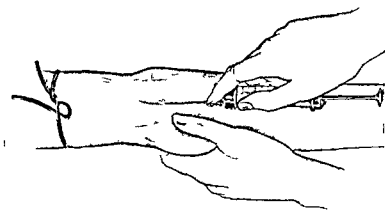


FIG 32

slowly there may be yawning, sneezing, or coughing just as consciousness is lost, but frequently the onset of unconsciousness is characterised simply by the appearance of generalised muscular relaxation, quiet shallow respiration and abolition of response to stimuli

It may be extremely difficult to differentiate between deep anaesthesia and light undisturbed anaesthesia. In both cases the respiration is shallow, there is general muscular relaxation and the eyeball is central. The depth of anaesthesia may not be revealed until a stimulus is applied. In deep anaesthesia respiration remains shallow whilst if anaesthesia is light the rate and excursion of respiration increase at once.

### Overdosage with Thiopentone

The immediate treatment of a gross overdose of any barbituric acid drug is the same as the treatment of overdose from any other anaesthetic. Artificial respiration (p. 327) must be continued until sufficient of the drug is excreted for the respiratory centre to recover its normal automatic activity.

The only analeptic drug of proven worth in the treatment of poisoning from barbituric acid derivatives is picrotoxin. This potent drug should be given intravenously at the rate of 1 mg per minute until the patient rouses or until the appearance of muscular twitchings, usually seen first in the face.

Where it is desired merely to accelerate the rate of recovery from thiopentone anaesthesia Coramine in doses of 5 c.c. intravenously or intramuscularly will be found effective.

### REFERENCES

- (1) *British Pharmacopæia* (7th Addendum) 1932 65
- (2) Burstein C. L. 1937 *Proc Soc exp Biol* **37**, 267
- (3) Burstein C. L. and Rovenstine E. A. 1939 Personal communication
- (4) *Gray's Anatomy* 1938 London 753

The advice usually given is that for a normal adult about 0.3 g (i.e. 3 c.c. of a 10 per cent solution) should be injected over 10-15 seconds, and then a pause made of 30 seconds the average time for the circulation of the blood so that the effect of this amount can be noted before a decision is taken as to what additional amount if any should be given.

The rate of injection has a very important influence on the effect which a given quantity of thiopentone will produce (p. 26). Slow injection results in a widespread distribution of the drug throughout the body whereas rapid injection produces an immediate knock out effect on the brain after which the more widespread distribution takes place. Thus although 0.6 g injected uniformly over a period of say two minutes may not produce unconsciousness but only drowsiness or drunkenness lasting for some considerable time 0.3 g to the same patient injected as rapidly as possible will produce transient deep anæsthesia followed within a few minutes by almost complete recovery.

The rate of excretion of thiopentone also influences the rate of attainment and the duration of anæsthesia. Excretion appears to be more rapid in the robust individual.

The effect of a given quantity of thiopentone therefore depends on the rate of injection, the amount injected, the rate of excretion of the drug and on the resistance of the patient to the effects of narcotics generally.

Cases are reported where it is alleged that after thiopentone given intravenously the development of anæsthesia was delayed. According to such reports although the injection had no immediate effect it was followed some hours later by drowsiness. There can be no doubt that a large part if not the whole of the injected solution had been deposited extravascularly. This explanation has often been confirmed by the development within twenty-four hours of pain and sometimes of inflammation at the site of injection. If the drug is given wholly into a vein and the patient does not become unconscious within a minute this is proof that it has been given either too slowly or in insufficient dosage.

### Signs of Anæsthesia with Thiopentone

When the injection is made rapidly the first and second stages of anæsthesia are passed through almost instantaneously. The patient, from talking quite rationally stops suddenly perhaps in the middle of a sentence and the next second a gentle snore or the automaticity of respiration shows that he is anæsthetised. If the injection is made

direction in which the nozzle is aimed Ethyl chloride burns with a bright blue flame but it can be used safely in a room with an open flame

The liquid can be purchased mixed with 0.5 per cent eau de Cologne Although ethyl chloride vapour has not an objectionable smell the addition of the eau de Cologne makes induction more pleasant

Ethyl chloride is a potent anæsthetic agent Its advantages are

- 1 It is not unpleasant to inhale
- 2 It is easy to administer
- 3 It is economical
- 4 It is easily portable
- 5 Induction of anæsthesia is rapid
- 6 The signs of anæsthesia are simple and well defined
- 7 Recovery after short anæsthesia is rapid

The disadvantages of ethyl chloride are

1 Nausea, vomiting headache and prostration frequently occur after deep or prolonged narcosis Were it not for this disadvantage, ethyl chloride would be used more extensively than it is at present

2 The stages of anæsthesia are passed through so rapidly that it is easy to administer an overdose For the same reason it is an unsuitable drug for the maintenance of anæsthesia

3 Involuntary micturition and even defæcation are more liable to occur than under anæsthesia with other agents

### Indications for the Use of Ethyl Chloride

Although potentially highly dangerous, ethyl chloride used intelligently is a safe anæsthetic That its administration needs the strictest supervision does not detract from its value A patient fit enough to be anaesthetised by any other anæsthetic can be given ethyl chloride The special indications for its use are

1 As a sole anæsthetic for operations of short duration particularly dental extractions in children The margin of safety here is high and is not exceeded by any other anæsthetic except possibly vinesthene

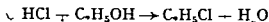
2 As a supplementary anæsthetic to nitrous oxide anæsthesia for young children or resistant adults including alcoholics

3 To provide a quick induction when prolonged anæsthesia is to be maintained by ether Its use for this purpose has been largely

## CHAPTER XVI

### ETHYL CHLORIDE

ETHYL CHLORIDE ( $C_2H_5Cl$ ) was first used as an anæsthetic in 1848<sup>1</sup>. It is usually prepared by passing gaseous hydrochloric acid into boiling ethyl alcohol



The yield is greatly improved in the presence of anhydrous zinc chloride which acts as a catalyst (Grove's process 1874). It may also be prepared by the reaction between ethylene gas and gaseous hydrochloric acid at high temperatures ( $130^\circ C$ )



The boiling point of ethyl chloride is  $12.4^\circ C$ . At ordinary room temperature and pressure it is therefore a gas but the increase of pressure required to liquefy it is so small that it can be kept with safety as a liquid in glass containers. It is supplied in bottles of a convenient size and shape from which it can escape through a nozzle when the small control lever is depressed. If the nozzle is directed downwards opening it by means of the lever will cause the expulsion of a jet of liquid ethyl chloride which rapidly volatilises. In this way the anæsthetic agent can be directed on to a mask at a reasonable concentration with very little loss. An attempt to obtain a spray when the nozzle is directed upwards releases only the ethyl chloride which has already vaporised above the fluid in the bottle so that the rate of ejection of the

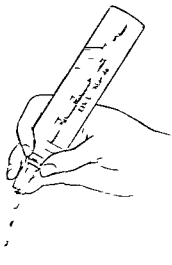


FIG. 37—Bottle containing 60 c.c. of ethyl chloride. Note the pointed nozzle and convenient control.

anæsthetic is negligible and since the vapour density of ethyl chloride gas is 2.2 any which does escape falls downwards i.e. away from the

direction in which the nozzle is aimed Ethyl chloride burns with a bright blue flame but it can be used safely in a room with an open flame

The liquid can be purchased mixed with 0.5 per cent eau de Cologne Although ethyl chloride vapour has not an objectionable smell the addition of the eau de Cologne makes induction more pleasant

Ethyl chloride is a potent anæsthetic agent Its advantages are

- 1 It is not unpleasant to inhale
- 2 It is easy to administer
- 3 It is economical
- 4 It is easily portable
- 5 Induction of anæsthesia is rapid
- 6 The signs of anæsthesia are simple and well defined
- 7 Recovery after short anæsthesia is rapid

The disadvantages of ethyl chloride are

1 Nausea, vomiting headache and prostration frequently occur after deep or prolonged narcosis Were it not for this disadvantage, ethyl chloride would be used more extensively than it is at present

2 The stages of anæsthesia are passed through so rapidly that it is easy to administer an overdose For the same reason it is an unsuitable drug for the maintenance of anæsthesia

3 Involuntary micturition and even defæcation are more liable to occur than under anæsthesia with other agents

### Indications for the Use of Ethyl Chloride

Although potentially highly dangerous ethyl chloride used intelligently is a safe anæsthetic That its administration needs the strictest supervision does not detract from its value A patient fit enough to be anæsthetised by any other anæsthetic can be given ethyl chloride The special indications for its use are

1 As a sole anæsthetic for operations of short duration particularly dental extractions in children The margin of safety here is high and is not exceeded by any other anæsthetic except possibly vinesthene

2 As a supplementary anæsthetic to nitrous-oxide anæsthesia for young children or resistant adults including alcoholics

3 To provide a quick induction when prolonged anæsthesia is to be maintained by ether Its use for this purpose has been largely



superseded in recent years by that of basal anæsthetics such as avertin or intravenous anæsthetics such as thiopentone

### Signs of Anæsthesia with Ethyl Chloride

The stages of ethyl chloride anæsthesia can be conveniently divided into three induction surgical anæsthesia and overdose Forunately these stages are readily distinguishable, and since ethyl chloride is employed only for short operations no subdivisions are necessary The character of the respiration is the guide *par excellence* to the stage of anæsthesia

Induction is often peaceful but breath holding or other irregularity of respiration screaming or tightening of the body musculature are not uncommon Breath holding may occur early in induction either because the child is unco operative or because the strength of the vapour on the mask is increased too quickly Just before the establishment of surgical anæsthesia there is usually a tautening of the muscles of the body This is responsible for a hesitancy in respiration which may simulate breath holding

The stage of surgical anæsthesia is marked by regularity of respiration relaxation of the muscles of the limbs and flushing of the face If induction has not been peaceful the transition from irregular breathing to the automatic respirations of surgical anæsthesia is often dramatic and is more noticeable with ethyl chloride than with any other anæsthetic With thiopentone and generally with nitrous oxide the onset of the automatic respirations of surgical anæsthesia is seldom striking because the patient breathes quietly and regularly from the beginning Respiration at this stage of anæsthesia with ethyl chloride is regular slightly more rapid than normal and moderately deep and audible The breathing resembles that of a child sleeping soundly

The eye signs are unreliable A large pupil may be seen at any stage of anæsthesia Strabismus is not uncommon during light anæsthesia

In the stage of overdose respiration becomes progressively shallower This is accompanied by pallor and an increase in muscular flaccidity and by wide dilatation of the pupil As with all other anæsthetics excepting chloroform occasionally respiratory failure precedes cardiac failure though in the case of ethyl chloride only by a short interval

### Dangers of Ethyl Chloride and their Avoidance

When any anæsthetic is given by the open method, anæsthesia deepens for a few breaths after the mask is removed because of the

absorption of the gas still in the lungs. This effect is particularly noticeable with ethyl chloride because of its potency. Therefore if removal of the mask is delayed until anaesthesia is deep, it will be dangerously deep a few seconds later. For this reason until the administrator is experienced it is a good rule immediately regular respiration is established to remove the mask and allow one breath of air before the operation is commenced

Early in induction an unco-operative child may hold his breath long enough for slight cyanosis to be produced before the accumulating carbon dioxide forces a deep breath. If the mistake is made of spraying ethyl chloride on to the mask when the patient is not breathing a strongly concentrated vapour accumulates and a single deep inspiration of this strong vapour might lead to respiratory paralysis. The observance of the rule if the patient holds his breath stop the anaesthetic will prevent the occurrence of this emergency.

Although some degree of respiratory irregularity is common during induction it is not invariable. Some patients notably the good type of child breathe shallowly and regularly throughout. The muscles are not tautened nor the breath held at any stage, so that the onset of muscular relaxation and the automatic respiration of surgical anaesthesia may be overlooked. If the anaesthetist, under the impression that the child is not yet 'off' continues to give ethyl chloride after anaesthesia is established dangerously deep anaesthesia will quickly result.

Respiratory obstruction is a grave danger in ethyl chloride anaesthesia. Because of the potency of the agent obstruction may be followed quickly by grave tissue anoxia. Cessation of respiratory effort at any time must be immediately investigated. The causes are (i) voluntary breath holding in an unco-operative patient (ii) reflex breath holding from too strong a concentration of vapour (iii) mechanical obstruction of respiration and (iv) gross overdosage. These are generally easy to differentiate but if there is any doubt, treatment for mechanical obstruction and overdosage must be instituted.

During induction with ethyl chloride spasm of the laryngeal muscles may cause inspiratory stridor which, unlike that developing with other anaesthetics may persist into deep anaesthesia. Attempts are sometimes made to deepen anaesthesia with the object of relaxing the laryngeal muscles but such attempts are dangerous because the spasm may not relax until anaesthesia is dangerously deep.

When stridor occurs the mask should be removed to allow the patient to breathe air. The stridor disappears before consciousness is regained and if the mask is then reapplied and anaesthesia gradually

deepened, it rarely returns If it does recur, the administration should be discontinued and no attempt should be made to extract until the stridor ceases Respiratory exchange is already markedly affected by the obstruction which causes the stridor and if to this further respiratory obstruction is added, e.g. by the sponge or by the dentist pushing the lower jaw and tongue backwards, the limitation of oxygen intake will increase to a dangerous degree the tissue anoxia already existing After the stridor passes off approximately thirty seconds operating time remains before the patient recovers The cause of stridor in these cases has not been satisfactorily explained The fact that it occurs with less frequency now than formerly has been attributed to greater purity of the drug but this cannot be a complete explanation for ethyl chloride out of the same bottle may produce stridor in one patient and uneventful anæsthesia in another

Pallor during administration of ethyl chloride is a danger sign Ethyl chloride causes dilatation of the peripheral vessels giving the patient a flushed appearance and absence of this normal reaction is of serious significance In some cases pallor occurs only after a large quantity has been administered but in children with an idiosyncrasy it is seen relatively early In such children soon after the loss of consciousness the respiratory excursion diminishes rapidly and the colour which probably has never been good becomes still paler Muscular flaccidity return of the eccentric eyeball to centre and wide dilatation of the pupil occur coincidentally with the pallor but are more easily overlooked When pallor occurs it is wise to suspend the operation until anæsthesia has lightened Further administration of ethyl chloride or the existence of any respiratory obstruction at this stage is fraught with great danger

Since small doses of ethyl chloride produce rapid changes in the depth of anæsthesia the principal danger in its use is that of over dosage for signs of which the anæsthetist must be constantly on the watch Close adherence to the following rules will make for safety

**1 A free airway must be maintained**—This applies to all anæsthetised patients but is particularly to be stressed when powerful drugs are used to produce unconsciousness

**2 Every breath should be seen or heard**—Generally either inspiration or expiration is audible and the expired air after passing through the frozen mask is always visible The respiratory excursions of the chest can usually be followed with ease but it must be remembered that these movements are not an infallible indication of the expansion of the lungs If respiration is obstructed and unconscious

ness is not deep movement of the thoracic wall may continue for a short period without achieving any pulmonary ventilation

3 If the patient holds his breath, stop the anæsthetic until the cause of cessation of respiration is discovered and until breathing recommences

4 If there is any doubt about the depth of anæsthesia the patient must be treated as if deeply anæsthetised until the contrary is proved

## ADMINISTRATION

Ethyl chloride may be given by the open or closed method Although the former is more wasteful we prefer it mainly because it is more pleasant for the patient and partly because the oxygen intake is not restricted Before discussing the respective advantages of these alternative procedures, we shall consider certain points common to them both

Two bottles of ethyl chloride should be at hand before induction is begun This precaution is advisable in case one becomes empty or falls and breaks or in case the nozzle becomes frozen and the size of the spray is thereby so much reduced that induction is greatly delayed

An attempt must be made to insert a prop in a child's mouth before the anæsthetic is started, and efforts to spit it out should be frustrated by the application of pressure with the little finger under the chin

If the administration is stopped as soon as respiration becomes automatic, about 60 seconds of anæsthesia will be obtained, it is followed by analgesia lasting 20 to 30 seconds Complete recovery is not so rapid as after nitrous oxide and the patient may remain dazed for a minute or two On recovery the previously flushed face often becomes pale and this especially if the pallor is of the circumoral type suggests that vomiting is imminent

**Repeated applications of ethyl chloride**—In dealing with children if lengthy extractions are to be made it has to be decided whether to attempt to complete the work on a single occasion or to divide it between two or more sittings In making the decision, it must be remembered that children even more than adults dislike repeated anæsthetics If the former course is chosen there are still alternatives the more popular is to secure a sufficient duration of anæsthesia by producing profound unconsciousness by a single administration of a large enough dose of ethyl chloride Most

anæsthetists have a strong prejudice against the other alternative of reapplying ethyl chloride during dental work. It outweighs for them the danger of overdosage which accompanies the attempt to secure long anæsthesia from one application. However even when this risk is taken a sufficiently prolonged anæsthesia cannot be guaranteed. If the operation proves longer than was anticipated it may have to be abandoned because of returning consciousness and completed at a further sitting. Moreover the complications of vomiting and prostration are associated more with depth than with length of anæsthesia.

For these reasons we believe that the better course is to make repeated small applications during a single sitting. It is often convenient to give only enough ethyl chloride to allow one half of the mouth to be operated on and then before the child is conscious to apply an additional amount sufficient to allow extractions on the other side to be completed. Readministration should not be deferred so long that the child is responsive enough to struggle for he is then liable to pull the mask away from his face. There may then be difficulty in settling him down again and his movements favour aspiration of any foreign body which may have been overlooked in the mouth. The anæsthetist must therefore stop the dentist operating at the point where he thinks recovery and restlessness will take place in a few seconds. The signs of returning consciousness are movements of the body or limbs, phonation, screwing up of the face, and lachrymation. If reapplication of ethyl chloride is deferred until the patient is almost conscious vomiting may occur.

It is an inviolable rule that ethyl chloride or any other anæsthetic must never be reapplied unless the airway is clear and the mouth free from debris. The head should be tipped forward to drain the mouth of blood and the soiled sponge should be taken out and replaced by a clean one. If the fresh sponge becomes frozen during readministration of ethyl chloride it will cease to provide adequate protection and must again be changed before the operation is recommenced.

### Open Ethyl Chloride

The equipment for administering ethyl chloride by the open method is very simple. It consists merely of a Schimmelbusch mask (p. 175) and a few thicknesses of gauze. In case of necessity even these can be dispensed with, and an ordinary linen hand towel or bib held over the nose and mouth will make an effective substitute.

—This latter method although neat and always available has one disadvantage. The ethyl chloride coming into contact with the patient's face and lips and also with the operator's left hand is un-

pleasantly cold, and may, if applied long enough even cause a mild burn

A few drops of ethyl chloride are sprayed on the mask or hand towel, which is gradually lowered on to the patient's face Nothing is to be gained by a slow induction. The vapour concentration should be increased steadily and as rapidly as possible but the rate of increase should never be enough to make the patient uncomfortable nor to tempt him to hold his breath Many children can be persuaded to inhale the anæsthetic quietly, and a good anæsthetist may coax the patient to unconsciousness without upsetting the easy rhythm of normal respiration. A child may be disinclined to 'breathe in' ethyl chloride sprayed on the mask but he may often be persuaded



FIG 38



FIG 39

to blow away the scent. The hearty puffs are followed by proportionately deep inspirations, and anæsthesia quickly follows.

The unsympathetic anæsthetist may try to hurry induction by forcing a concentrated vapour suddenly on the child without waiting to see if he will co-operate in a more gradual induction. This inevitably results in some degree of breath holding or struggling. Even with a co-operative patient however induction though gradual should not be prolonged unduly.

Peaceful induction is impossible where the patient screams or struggles. If a child cries it is obvious that he is maintaining a good airway. Gradual increase of the dose should not then be attempted but ethyl chloride should be given freely to induce anæsthesia as quickly as possible. A spoilt obstreperous child is in this way easily and quickly subdued by ethyl chloride. The onset of surgical anæsthesia is here sudden and the mask must be removed immediately respiration becomes automatic.

Occasionally with nitrous oxide even with a co operative child it is advisable to supplement anæsthesia by the addition of ethyl chloride (p 127).

Rapidity of onset of anæsthesia is determined by the amount of ethyl chloride sprayed on the mask, and by the respiratory volume and rate. Variations in the respiratory rhythm make it impossible to give ethyl chloride by a rule-of thumb method. It is impossible to predict the quantity which will be needed both because patients vary so much



FIG 40 —Great Ormond Street method of giving ethyl chloride

in their requirements and because the amount wasted by evaporation cannot be estimated.

The amount of ethyl chloride used by the Great Ormond Street method is still less predictable. This technique introduced by Sington has however much to commend it. It is a leisurely method

in which the child himself is persuaded to hold the mask 8 to 10 inches away from his face while ethyl chloride is sprayed on it. He is allowed to sit up or lie down just as he chooses. The anaesthetist talks to him continuously, and by various manœuvres the mask is gradually lowered on to the face. This may take one or two minutes, and up to 30 c.c. of ethyl chloride may be sprayed on the mask before unconsciousness is reached. Such an induction is peaceful and well justifies the expenditure of extra time and material. Without realising what is happening the child becomes more and more stuporose, fails to answer questions and gradually allows the mask to be lowered on to his face.

For general surgery, a satisfactory alternative<sup>2</sup> to the Great Ormond Street method in a child who comes to the operating theatre conscious is to encourage him to "roll over on your side as if you're going to sleep." Once the child is in this natural position the mask is held a few inches away from his face and ethyl chloride sprayed on to it. Resistance is rare, the onset of unconsciousness is smooth, and the experience is not resented at the time or subsequently.

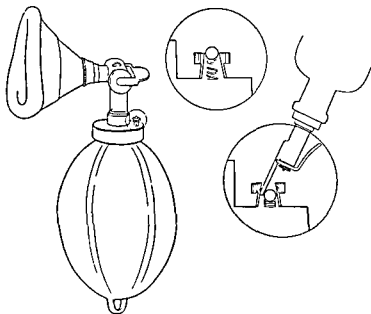


FIG. 41—Loosely's Inhaler

### Closed Ethyl Chloride

The chief advantage of the closed method is that it is economical. One argument frequently put forward in its favour is that since only a fixed amount is put into the bag and this amount is small, it is



Peaceful induction is impossible where the patient screams or struggles. If a child cries it is obvious that he is maintaining a good airway. Gradual increase of the dose should not then be attempted, but ethyl chloride should be given freely to induce anæsthesia as quickly as possible. A spoilt, obstreperous child is in this way easily and quickly subdued by ethyl chloride. The onset of surgical anæsthesia is here sudden and the mask must be removed immediately respiration becomes automatic.

Occasionally with nitrous oxide even with a co operative child it is advisable to supplement anæsthesia by the addition of ethyl chloride (p 127)

Rapidity of onset of anæsthesia is determined by the amount of ethyl chloride sprayed on the mask, and by the respiratory volume and rate. Variations in the respiratory rhythm make it impossible to give ethyl chloride by a rule of thumb method. It is impossible to predict the quantity which will be needed both because patients vary so much



FIG 40 —Great Ormond Street method of giving ethyl chloride

in their requirements and because the amount wasted by evaporation cannot be estimated

The amount of ethyl chloride used by the Great Ormond Street method is still less predictable. This technique introduced by Sington has however much to commend it. It is a leisurely method

patient unless the asphyxial element which must be introduced either makes operating difficult or is enough to jeopardise the patient's safety.

Ethyl chloride is still of value, however, as a supplementary agent in cases where it is discovered, after nitrous oxide anaesthesia has been embarked upon, that a patient of powerful physique cannot be settled to a suitable level of anaesthesia. It may also prove useful when a tooth breaks unexpectedly during extraction from such an individual. After some minutes during which the operative stimulus may have to be increased the patient is likely to become progressively difficult to control and the addition of a little ethyl chloride is remarkably effective in establishing peaceful anaesthesia and facilitating its subsequent maintenance.

The nose-piece is retained in position and the mouth covered by a hand towel or the upturned bib on to which ethyl chloride is sprayed. If the patient is breathing mainly through his mouth anaesthesia will be produced almost entirely by the ethyl chloride but as it deepens he will resort to nose breathing and the anaesthetic is then continued with nitrous oxide and oxygen. The question whether the patient can be exposed safely to an appreciable degree of oxygen-hack or not



FIG 43

depends on the amount of ethyl chloride given. Cyanosis should not be allowed to occur after a moderate amount of ethyl chloride but if the amount given has been small a slight reduction in the oxygen intake may be necessary in order to maintain tranquil anaesthesia with nitrous oxide.

difficult to give an overdose, except by a gross error of judgment in estimating the amount needed

On the bag holder is a small aperture guarded by a ball which is kept in position by a spring. This ball is easily depressed by the nozzle of the container from which ethyl chloride then can be sprayed into the bag. At first the mask is applied intermittently so that the patient inspires from the air and expires into the bag thus inflating it. After the bag is full it is held firmly on the face to prevent any leakage. According to the size and resistance of the patient from 2 to 6 c c of ethyl chloride are then gradually sprayed into the bag. As soon as anæsthesia is established it will not be deepened by retention of the mask on the face since practically all the ethyl chloride is already absorbed from the bag and in the early stages is distributed largely to the brain producing deep anæsthesia. If the mask is kept to the face valuable operating time is lost during which the anæsthetic is merely being redistributed from the brain to the tissues with consequent lessening of the degree of unconsciousness.



FIG. 42

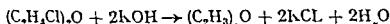
### Ethyl Chloride as a Supplement to Nitrous oxide Anæsthesia

Within recent years a small preliminary intravenous injection of thiopentone or hexobarbitone given to the robust adult patient before continuing with nitrous oxide has in many cases replaced the supplementary ethyl chloride which might otherwise be indicated for example where nitrous oxide is not strong enough to control the

## CHAPTER XVII

## VINESTHENE

DI VINYLETHER ( $C_2H_5O$ ) is less stable than diethyl ether and is very susceptible to the action of oxygen, heat, and light. Vinesthene is a proprietary brand of di vinyl ether to which has been added 0.01 per cent phenyl  $\alpha$  naphthylamine and 4 per cent absolute alcohol to prevent deterioration. The liquid is marketed in dark glass bottles to give added protection. Di vinyl ether is prepared from a chlorine derivative of diethyl ether di( $\beta$  chloroethyl) ether by heating it with solid potassium hydroxide at  $200^\circ C$ .



The use of vinesthene as an anæsthetic was first suggested by Leake<sup>1</sup>. It was introduced into clinical anæsthesia in 1933<sup>2</sup>. It is an almost colourless, volatile highly inflammable liquid with a specific gravity of 0.8, and it boils at  $29^\circ C$ . The vapour density of the gas is 2.4. It has a characteristic petrol like smell and mixtures with air or oxygen are highly explosive.

It is potent and induces anæsthesia rapidly and not unpleasantly. It is useful in general surgery either merely as an induction agent or as the sole anæsthetic for short operations. It has a particular usefulness for short operations and has become especially popular for dental surgery in children. For more prolonged operations mixtures with ethyl ether in any proportions are satisfactory for the maintenance of anæsthesia and a mixture containing 25 per cent vinesthene and 75 per cent ethyl ether is much in favour both for induction and maintenance. Vinesthene has been used with success in midwifery both for analgesia and to obtain anæsthesia at the end of the second stage of labour.

## Vinesthene for Dental Surgery

Vinesthene is now often used where ethyl chloride would formerly have been given. The indications for its use are very similar to those for ethyl chloride. Either drug can be given by the open or by the closed method. We consider that the open method is more humane and comparing the two drugs given by the open method we find ethyl chloride to be more convenient, more rapid, and much more

In case of extreme violence the nose piece also must be replaced by the bib and induction completed with ethyl chloride alone. A struggling man breathes deeply so that anæsthesia is induced quickly. As soon as he becomes tranquil anæsthesia is continued nasally with nitrous oxide.

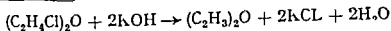
#### REFERENCES

- (1) Heyfelder F 1848 Die Versuche mit dem Schwefeläther Salzäther und Chloroform Erlangen 83-85
- (2) Bannister F B and Scott R D 1942 Personal communication

## CHAPTER XVII

## VINESTHENE

DI VINYL ETHER  $(C_2H_3)_2O$  is less stable than diethyl ether and is very susceptible to the action of oxygen, heat and light. Vinesthene is a proprietary brand of di vinyl ether to which has been added 0.01 per cent phenyl  $\alpha$  naphthylamine and 4 per cent absolute alcohol to prevent deterioration. The liquid is marketed in dark glass bottles to give added protection. Di vinyl ether is prepared from a chlorine derivative of diethyl ether, di( $\beta$ -chloroethyl) ether by heating it with solid potassium hydroxide at  $200^\circ C$ .



The use of vinesthene as an anæsthetic was first suggested by Leake<sup>1</sup>. It was introduced into clinical anæsthesia in 1933<sup>2</sup>. It is an almost colourless volatile highly inflammable liquid with a specific gravity of 0.8 and it boils at  $29^\circ C$ . The vapour density of the gas is 2.4. It has a characteristic petrol like smell, and mixtures with air or oxygen are highly explosive.

It is potent, and induces anæsthesia rapidly and not unpleasantly. It is useful in general surgery either merely as an induction agent or as the sole anæsthetic for short operations. It has a particular usefulness for short operations and has become especially popular for dental surgery in children. For more prolonged operations mixtures with ethyl ether in any proportions are satisfactory for the maintenance of anæsthesia, and a mixture containing 25 per cent vinesthene and 75 per cent ethyl ether is much in favour both for induction and maintenance. Vinesthene has been used with success in midwifery both for analgesia and to obtain anæsthesia at the end of the second stage of labour.

## Vinesthene for Dental Surgery

Vinesthene is now often used where ethyl chloride would formerly have been given. The indications for its use are very similar to those for ethyl chloride. Either drug can be given by the open or by the closed method. We consider that the open method is more humane and comparing the two drugs given by the open method we find ethyl chloride to be more convenient more rapid and much more

In case of extreme violence the nose piece also must be replaced by the bib and induction completed with ethyl chloride alone. A struggling man breathes deeply so that anæsthesia is induced quickly. As soon as he becomes tranquil anæsthesia is continued nasally with nitrous oxide.

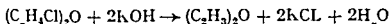
#### REFERENCES

- (1) Heyfelder F 1848 Die Versuche mit dem Schwefelather Salzather und Chloroform Erlangen 83-85
- (2) Bannister F B and Scott R D 1942 Personal communication

## CHAPTER XVII

## VINESTHENE

DI VINYL ETHER ( $C_4H_6$ )O is less stable than diethyl ether and is very susceptible to the action of oxygen, heat and light. Vinesthene is a proprietary brand of di vinyl ether to which has been added 0.01 per cent phenyl- $\alpha$  naphthylamine and 4 per cent absolute alcohol to prevent deterioration. The liquid is marketed in dark glass bottles to give added protection. Di vinyl ether is prepared from a chlorine derivative of diethyl ether di( $\beta$ -chloroethyl) ether by heating it with solid potassium hydroxide at  $200^\circ C$ .



The use of vinesthene as an anæsthetic was first suggested by Leake<sup>1</sup>. It was introduced into clinical anæsthesia in 1933<sup>2</sup>. It is an almost colourless volatile highly inflammable liquid with a specific gravity of 0.8 and it boils at  $29^\circ C$ . The vapour density of the gas is 2.4. It has a characteristic petrol like smell and mixtures with air or oxygen are highly explosive.

It is potent and induces anæsthesia rapidly and not unpleasantly. It is useful in general surgery either merely as an induction agent or as the sole anæsthetic for short operations. It has a particular usefulness for short operations and has become especially popular for dental surgery in children. For more prolonged operations mixtures with ethyl ether in any proportions are satisfactory for the maintenance of anæsthesia and a mixture containing 25 per cent vinesthene and 75 per cent ethyl ether is much in favour both for induction and maintenance. Vinesthene has been used with success in midwifery both for analgesia and to obtain anæsthesia at the end of the second stage of labour.

### Vinesthene for Dental Surgery

Vinesthene is now often used where ethyl chloride would formerly have been given. The indications for its use are very similar to those for ethyl chloride. Either drug can be given by the open or by the closed method. We consider that the open method is more humane, and comparing the two drugs given by the open method we find ethyl chloride to be more convenient more rapid and much more



In case of extreme violence the nose piece also must be replaced by the bib and induction completed with ethyl chloride alone. A struggling man breathes deeply so that anæsthesia is induced quickly. As soon as he becomes tranquil anæsthesia is continued nasally with nitrous oxide.

#### REFERENCES

- (1) Heyfelder, F. 1848 Die Versuche mit dem Schwefeläther Salzäther und Chloroform Erlangen 83-85
- (2) Bannister F. B. and Scott R. D. 1942 Personal communication

The mask is then held firmly to the face. The disadvantage of this inhaler is that the strength of the vapour cannot be increased gradually. This is to some extent offset by the fact that a few breaths suffice to produce anaesthesia but before this is reached the sudden irritation from the concentrated vapour usually leads to crying and physical resistance during the induction. Hence the breathing is at first irregular, but in most cases if no vapour is lost by leakage the automatic respiration characteristic of surgical anaesthesia soon sets in. Occasionally however the irritating vapour causes laryngeal spasm, and the anoxæmia from continuous re-breathing may result in jerky irregular breathing. In either of these cases the mask should be removed and the patient allowed one or two breaths of air to relieve the condition before extractions begin.

It is essential to retain the mask firmly on the face to prevent any outward escape of vinylsthene. Usually anaesthesia is established in about one minute. The mask should then be removed and extractions commenced at once. As described in the chapter on ethyl chloride if the mask is kept too long applied the anaesthesia becomes lighter. The depth and duration of anaesthesia depend upon the dose introduced into the inhaler on whether any is lost by leakage and upon the size

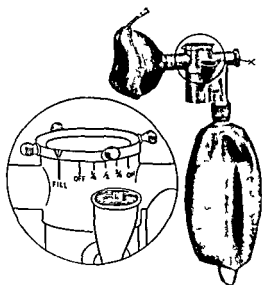


FIG. 45 —The Oxford Inhaler

and resistance of the patient. Minor variations in the anaesthesia occur from patient to patient but on an average 45–60 seconds of tranquil anaesthesia is provided and after this recovery of consciousness is abrupt.

economical Although in these respects ethyl chloride is more satisfactory vinesthene has two overriding advantages, and we frequently administer it even by the closed method in preference to ethyl chloride by the open method These advantages are

1 A striking absence of undesirable after effects such as post anæsthetic vomiting and collapse after short operations the patient is able to leave the surgery within a minute or two apparently none the worse for his experience

2 Less masseteric spasm so that the mouth can be opened with comparative ease in cases where the prop has been spat out during induction

### Signs of Anæsthesia

The crying and struggling common in the early induction period when this drug is used stop after a few breaths and respiration then becomes regular Even if it is regular from the beginning the automatic character indicating surgical anæsthesia is usually easily recognisable General muscular relaxation occurs at the same time the eyeballs become fixed usually eccentrically and the conjunctival reflex disappears When given by the closed method the depletion of the oxygen in the bag of the inhaler due to continued rebreathing, may cause slight cyanosis

### Administration by the Closed Method

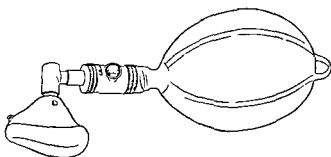


FIG 44 —Goldman's Inhaler

*Goldman's Inhaler* —The barrel contains a sponge on to which the content of a vinesthene ampoule (3 c c or 5-c c ampoules are available) is poured The dose generally recommended for children of all ages is 3 c c but for those above the age of 5 we prefer to use 5 c c A prop is inserted and the bag is distended either by the patient's expiration after the face mask has been applied at the end of an inspiration or it is blown up by the anæsthetist

The mask is then held firmly to the face. The disadvantage of this inhaler is that the strength of the vapour cannot be increased gradually. This is to some extent offset by the fact that a few breaths suffice to produce anaesthesia but before this is reached the sudden irritation from the concentrated vapour usually leads to crying and physical resistance during the induction. Hence the breathing is at first irregular but in most cases if no vapour is lost by leakage the automatic respiration characteristic of surgical anaesthesia soon sets in. Occasionally however the irritating vapour causes laryngeal spasm, and the anoxæmia from continuous rebreathing may result in jerky irregular breathing. In either of these cases the mask should be removed and the patient allowed one or two breaths of air to relieve the condition before extractions begin.

It is essential to retain the mask firmly on the face to prevent any outward escape of vinesthene. Usually anaesthesia is established in about one minute. The mask should then be removed and extractions commenced at once. As described in the chapter on ethyl chloride if the mask is kept too long applied the anaesthesia becomes lighter. The depth and duration of anaesthesia depend upon the dose introduced into the inhaler on whether any is lost by leakage and upon the size

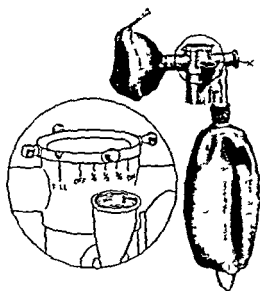


FIG. 45.—The Oxford Inhaler

and resistance of the patient. Minor variations in the anaesthesia occur from patient to patient but on an average 45–60 seconds of tranquil anaesthesia is provided and after this recovery of consciousness is abrupt.

economical Although in these respects ethyl chloride is more satisfactory vinesthene has two overriding advantages and we frequently administer it even by the closed method in preference to ethyl chloride by the open method These advantages are

1 A striking absence of undesirable after effects such as post anæsthetic vomiting and collapse after short operations the patient is able to leave the surgery within a minute or two apparently none the worse for his experience

2 Less masseteric spasm so that the mouth can be opened with comparative ease in cases where the prop has been spat out during induction

### Signs of Anæsthesia

The crying and struggling common in the early induction period when this drug is used stop after a few breaths and respiration then becomes regular Even if it is regular from the beginning the automatic character indicating surgical anæsthesia is usually easily recognisable General muscular relaxation occurs at the same time the eyeballs become fixed, usually eccentrically and the conjunctival reflex disappears When given by the closed method the depletion of the oxygen in the bag of the inhaler due to continued rebreathing may cause slight cyanosis

### Administration by the Closed Method

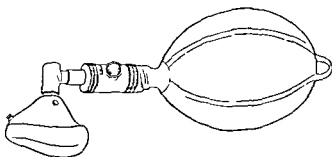


FIG 44 —Goldman's Inhaler

*Goldman's Inhaler* —The barrel contains a sponge on to which the content of a vinesthene ampoule (3-c c or 5-c c ampoules are available) is poured The dose generally recommended for children of all ages is 3 c c but for those above the age of 5 we prefer to use 5 c c A prop is inserted and the bag is distended either by the patient's expiration after the face mask has been applied at the end of an inspiration or it is blown up by the anæsthetist

The mask is then held firmly to the face. The disadvantage of this inhaler is that the strength of the vapour cannot be increased gradually. This is to some extent offset by the fact that a few breaths suffice to produce anaesthesia, but before this is reached the sudden irritation from the concentrated vapour usually leads to crying and physical resistance during the induction. Hence the breathing is at first irregular, but in most cases if no vapour is lost by leakage the automatic respiration characteristic of surgical anaesthesia soon sets in. Occasionally, however, the irritating vapour causes laryngeal spasm, and the anoxaemia from continuous rebreathing may result in jerky irregular breathing. In either of these cases the mask should be removed and the patient allowed one or two breaths of air to relieve the condition before extractions begin.

It is essential to retain the mask firmly on the face to prevent any outward escape of vinylene. Usually anaesthesia is established in about one minute. The mask should then be removed and extractions commenced at once. As described in the chapter on ethyl chloride if the mask is kept too long applied, the anaesthesia becomes lighter. The depth and duration of anaesthesia depend upon the dose introduced into the inhaler on whether any is lost by leakage and upon the size

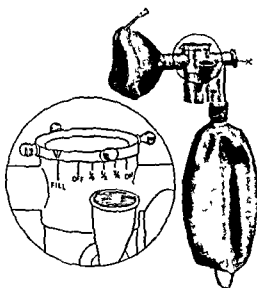


FIG. 45—The Oxford Inhaler

and resistance of the patient. Minor variations in the anaesthesia occur from patient to patient but on an average 45-60 seconds of tranquil anaesthesia is provided and after this recovery of consciousness is abrupt.

economical Although in these respects ethyl chloride is more satisfactory *vinesthene* has two overriding advantages and we frequently administer it even by the closed method in preference to ethyl chloride by the open method These advantages are

1 A striking absence of undesirable after effects such as post anæsthetic vomiting and collapse after short operations the patient is able to leave the surgery within a minute or two, apparently none the worse for his experience

2 Less masseteric spasm so that the mouth can be opened with comparative ease in cases where the prop has been spat out during induction

### Signs of Anæsthesia

The crying and struggling common in the early induction period when this drug is used stop after a few breaths and respiration then becomes regular Even if it is regular from the beginning the automatic character indicating surgical anæsthesia is usually easily recognisable General muscular relaxation occurs at the same time the eyeballs become fixed usually eccentrically and the conjunctival reflex disappears When given by the closed method the depletion of the oxygen in the bag of the inhaler due to continued rebreathing may cause slight cyanosis

### Administration by the Closed Method

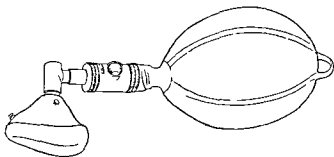


FIG 44 —Goldman's Inhaler

*Goldman's Inhaler* —The barrel contains a sponge on to which the content of a *vinesthene* ampoule (3 c c or 5-c c ampoules are available) is poured The dose generally recommended for children of all ages is 3 c c but for those above the age of 5 we prefer to use 5 c c A prop is inserted and the bag is distended either by the patient's expiration after the face mask has been applied at the end of an inspiration or it is blown up by the anæsthetist

The mask is then held firmly to the face. The disadvantage of this inhaler is that the strength of the vapour cannot be increased gradually. This is to some extent offset by the fact that a few breaths suffice to produce anaesthesia but before this is reached the sudden irritation from the concentrated vapour usually leads to crying and physical resistance during the induction. Hence the breathing is at first irregular but in most cases if no vapour is lost by leakage the automatic respiration characteristic of surgical anaesthesia soon sets in. Occasionally however, the irritating vapour causes laryngeal spasm, and the anoxemia from continuous re-breathing may result in jerky irregular breathing. In either of these cases the mask should be removed and the patient allowed one or two breaths of air to relieve the condition before extractions begin.

It is essential to retain the mask firmly on the face to prevent any outward escape of vinesthene. Usually anaesthesia is established in about one minute. The mask should then be removed and extractions commenced at once. As described in the chapter on ethyl chloride, if the mask is kept too long applied, the anaesthesia becomes lighter. The depth and duration of anaesthesia depend upon the dose introduced into the inhaler, on whether any is lost by leakage, and upon the size

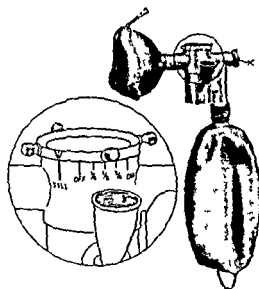


FIG. 45.—The Oxford Inhaler

and resistance of the patient. Minor variations in the anaesthesia occur from patient to patient but on an average 45–60 seconds of tranquil anaesthesia is provided and after this recovery of consciousness is abrupt.



*The Oxford Inhaler*<sup>3</sup> —This apparatus (Fig 45) modifies the Goldman inhaler in two respects which we consider are improvements (i) It contains a one way inlet valve ( $\Lambda$ ) for air which comes into action only if and when the bag is empty when it allows the patient to draw in supplementary air Expiration takes place entirely into the bag (ii) A by pass allows the concentration of anæsthetic vapour to be increased gradually

### Open Method



FIG 46



FIG 47

Vinesthene may be dropped on to a mask or on to a strip of lint hanging down over the nose The difficulty of obtaining a uniform rate of dropping from the bottle, and the expense of the drug have prevented a wider use of this method As with other anæsthetics the rate of administration is increased as rapidly as the patient will tolerate it If the supply is stopped at the onset of surgical anæsthesia the operating time afforded is about 60 seconds though it can safely be prolonged by delaying removal of the mask and continuing administration after automatic respiration is established

In comparison with the closed method induction takes rather longer and frequently 20 c c are necessary to establish anæsthesia

also salivation is more marked and may be troublesome. The duration of anæsthesia and of the period of recovery are dependent on the amount of vinesthene absorbed but generally they are both longer than when the drug is given by the closed method.



FIG 48



FIG 49

If desired anæsthesia by either method can be continued at a steady level by dropping vinesthene on to a folded strip of gauze held beneath the nostrils.

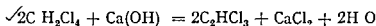
#### REFERENCES

- (1) Leale C D and Chen M Y 1930/31 *Proc Soc exp Biol* 28 151
- (2) Gelfan S and Bell I R 1930 *J Pharmacol* 47, 1
- (3) Boston T K and Salt R 1940 *Lancet* 2, 623

## CHAPTER XVIII

## TRICHLORETHYLENE (TRILENE)

TRICHLORETHYLENE ( $C_2HCl_3$ ) was popularised as a general anæsthetic by Hewer in 1941<sup>1</sup>. It can be prepared by treating acetylene gas ( $C_2H_2$ ) with chlorine to form tetrachlorethane ( $C_2H_2Cl_4$ ) which in turn is treated with dry calcium hydroxide to give trichlorethylene



Trichlorethylene under trade names such as Westrosol and Triklone has long been used commercially as a degreasing agent particularly in the dry cleaning of clothes and in removing oil from dismantled machinery but commercial preparations are not sufficiently pure for inhalation-anæsthesia. The compound is a colourless heavy liquid with a characteristic smell similar to but readily distinguishable from that of chloroform. The specific gravity is 1.47 and the boiling point 87°C. Pure trichlorethylene decomposes in strong sunlight. In a proprietary brand Trilene, 0.01 per cent of thymol is added to retard this reaction and the liquid is supplied in amber coloured bottles. A trace of waxoline blue is also added to give a distinctive colouring

## Indications for the Use of Trichlorethylene

Trichlorethylene vapour is non explosive in any conditions met with in clinical practice. It can therefore be used in the presence of a cautery or naked flame

Trilene used alone or to reinforce nitrous oxide oxygen anæsthesia is satisfactory for extra abdominal operations. Its use in abdominal surgery is limited by the inferior muscular relaxation obtained

It is non irritant to the mucous membrane of the respiratory tract, induction is therefore smooth and rapid after which, if desired, a change can be made to ether anæsthesia

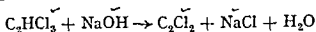
Trilene is especially valuable as an analgesic particularly in robust patients in whom good analgesia can be obtained without anoxia. Machines for the self administration of trilene air analgesia have been designed by Freedman<sup>2</sup> for midwifery and by Hill<sup>3</sup> for dentistry

### Mode of Administration

Trilene may be administered with nitrous oxide oxygen as the vehicle as for example when a Boyle's machine is used or with air drawn through Marrett's machine or the Oxford Vaporiser. Its use on an open mask is not practicable, because of the slow vaporisation at room temperature

### Decomposition with Soda-Lime

The chemical reaction between trilene and soda-lime produces dichloroacetylene



This reaction absolutely precludes the use of trilene in closed circuit anaesthesia. It takes place at blood heat and is accelerated by a rise of temperature such as occurs when  $\text{CO}_2$  is absorbed by soda-lime. Attention was drawn to this danger by a report of thirteen cases<sup>3</sup> two of them fatal of cranial nerve palsies due to the inhalation of decomposition products.

Trilene should never be left in a bottle attached to an apparatus which can be used for closed circuit anaesthesia. Further this agent should not be used for induction as a preliminary to closed circuit anaesthesia with ether because sufficient trilene vapour may be exhaled from the respiratory tract to allow the production of dichloroacetylene when the patient later breathes through the soda-lime canister.

### Tachypnoea

Muscular relaxation with trilene is not impressive. If the drug is pushed respiration may become rapid and shallow, and therefore inadequate. If a change is made to ether, respiration becomes normal and any desired relaxation is easily produced.

### Toxic Effects

Although the effect of trilene on metabolism is usually of no significance<sup>1</sup> two cases of liver necrosis resembling that seen in delayed chloroform poisoning have been reported.<sup>6,7</sup> There is no special danger to the heart from trilene<sup>1</sup> in spite of the fact that there are three chlorine atoms in its molecule as in chloroform. Sinus arrhythmia in light anaesthesia sometimes occurs in young patients but disappears when full anaesthesia is reached.

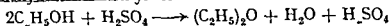
## REFERENCES

- (1) Hewer C L 1941 *Brit med J* 2 924
- (2) Freedman A 1943 *Lancet* 2 696
- (3) Hill B 1944 *Proc R Soc Med* 37 475
- (4) Marrett H R 1942 *Brit med J* 1 643
- (5) Humphrey J H and McClelland M 1944 *Brit med J* 2 315
- (6) Annotation 1944 *Lancet* 1 476
- (7) Herdman K N 1945 *Brit med J* 2 689

## CHAPTER XIX

## ETHER

ETHER (diethyl ether ( $C_2H_5)_2O$ ) is prepared by adding ethyl alcohol continuously to sulphuric acid at a temperature of about  $130^\circ C$ . An intermediate compound is formed, but the complete process is essentially the dehydration of ethyl alcohol



The ether so produced is purified by distillation

Ether first used as an anæsthetic just over one hundred years ago is still the safest and most popular anæsthetic for general use. It is noteworthy in general surgery that if an anæsthetist gets into difficulties with any other agent he almost invariably resorts to ether for help.

Ether is reliable and relatively non-toxic. It enables muscular relaxation adequate for an abdominal operation to be maintained without signs of respiratory depression and if an overdose is given, respiratory arrest occurs before the circulation is gravely impaired. Immediate removal of the mask and institution of artificial respiration will therefore, in all probability, result in recovery.

## PHYSICAL PROPERTIES

Ether is a colourless inflammable liquid with a specific gravity of 0.7 and a characteristic pungent odour. It may be mentioned here that an anæsthetist's first duty is to smell the content of the bottle of anæsthetic before he administers it to the patient. This precaution will eliminate the mistake frequently a fatal one of giving chloroform under the impression that it is ether. The boiling-point of ether ( $34.6^\circ C$ ) is just below body temperature and it volatilises readily at ordinary room temperature. Ether vapour has a density of 2.6 is very inflammable and with air or oxygen forms highly explosive mixtures. It should, therefore, never be used in the presence of a naked flame nor for an operation in which the cautery or diathermy is to be used. That ether vapour, two and a half times as heavy as air will sink must be borne in mind when positioning any apparatus from which a spark may be generated accidentally (e.g. an electrically operated suction pump).

## INDUCTION

The patient should be premedicated with atropine to prevent the salivation and bronchial secretion which otherwise frequently results

from the irritant action of ether vapour. If morphia is to be given it should be injected 1-2 hours before anæsthesia is due to begin (p 95)

Although ether is probably the most frequently used drug for the maintenance of anæsthesia it is seldom used from the beginning for induction is achieved more pleasantly and rapidly by such agents as ethyl chloride, vinylene, or thiopentone. If ether alone is to be used ample time must be allowed, since induction of anæsthesia is slow, particularly if the threshold of the respiratory centre to  $\text{CO}_2$  has been raised by morphia (p 47). Induction may take as long as 30 minutes and the anæsthetist must exercise considerable patience to avoid interrupting the regular rhythm of respiration. Ideally the ether should be given continuously and the concentration of vapour increased gradually yet as rapidly as the patient will tolerate it. An attempt to hasten induction by increasing too suddenly the concentration of vapour will defeat its own end since the oppressive vapour will check the regularity and the depth of respiration, and the onset of anæsthesia will be correspondingly delayed. The signs that the ether concentration is being increased too quickly are similar to the signs of irritation from any other cause on the unanæsthetised pharynx or larynx. There will be

(a) Swallowing, a protective reaction safeguarding the air passages or

(b) Breath-holding, which occurs coincidentally with swallowing or there may be breath holding without swallowing, or

(c) Coughing, or at a later stage

(d) Laryngeal spasm, causing inspiratory stridor and diminished pulmonary ventilation. Any of these reflex protective reactions retards the rate of absorption of ether, and should they occur the rate of administration must be reduced until a strength of vapour which can be tolerated is reached. When normal respiration recommences the anæsthetist must renew the attempt to find the maximum rate at which the vapour concentration can be increased without upsetting the respiratory rhythm.

When ether is to be used after an induction by ethyl chloride or thiopentone the optimum rate of administration is determined only by experience. If the ether is not given rapidly the patient will come round before enough has been absorbed to be effective, and if given too quickly laryngeal spasm may be precipitated. This troublesome complication of anæsthesia has many causes and during light ether anæsthesia may occur from irritation of the larynx by the direct action of strong ether vapour or by mucus secreted from the respiratory tract as a result of the strength of the vapour.

Provided respiration remains regular ether cannot be administered

too rapidly The beginner is advised to be cautious with all other anæsthetics but to be bold with ether, with which difficulties arise more frequently from under- than from over-dosage Provided a clear airway is maintained the patient will not collapse suddenly from an overdose Respiratory and other signs give ample warning of the approach of deep anæsthesia which can then be avoided by reducing the rate of administration The troubles of light ether anæsthesia are laryngeal spasm and vomiting The former is overcome as described by first decreasing and then progressively increasing the strength of vapour to which the larynx is exposed The latter may cause respiratory obstruction by the lodgment of a mass of food or acid regurgitation from the stomach by irritating the larynx may cause persistent laryngeal spasm which limits the intake of further anæsthetic and makes the attainment of surgical anæsthesia difficult

Although ether may be given by rectum mixed with olive oil or intravenously in saline, it is rarely administered other than by inhalation It may be given

- (a) By dropping it on an open mask, where it is vaporised
- (b) By delivering to the patient air oxygen or a mixture of nitrous oxide and oxygen which has been passed over or through ether contained in a bottle This is often referred to as the semi closed method
- (c) In a closed circuit
- (d) By the Oxford Vaporiser (pp 347-355)

### Open drop Method

The mask is covered with about a dozen thicknesses of gauze

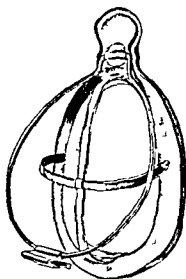
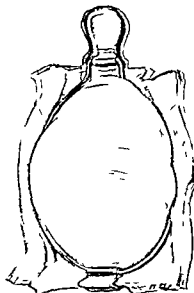


FIG  
50

FIG  
51

Schimmelbusch  
Mask





which are held in position by the spring band The gauze must not be thick enough to interfere with free respiration

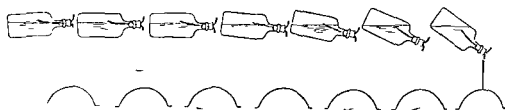


FIG 52 (After Waters)

Provided free regular respiration is not interrupted, the rate of flow of ether on to the mask must be continuous and must be increased uniformly

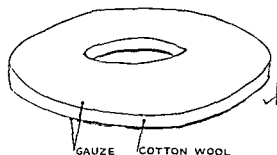


FIG 53

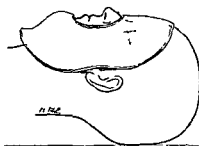


FIG 54



FIG 55

A gamgee pad—i.e. cotton wool covered with gauze—is a help to the efficient administration of ether by the open drop method

The concentration of ether vapour can be increased by inserting a gamgee pad underneath the mask thus preventing the patient from breathing any air which does not pass through the ether laden mask

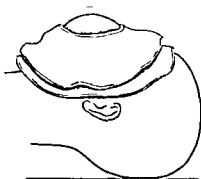


FIG 56

The concentration of anæsthetic vapour can be still further increased by covering the mask with another gamgee pad

### "Semi-closed" Method

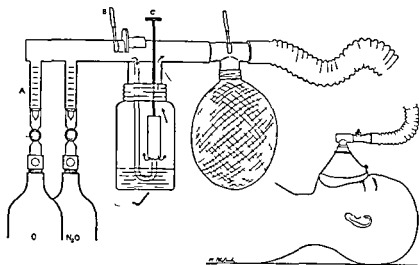


FIG 57

The rates of flow of the selected gases (usually nitrous oxide and oxygen) are measured by flowmeters (A) and can be controlled by opening or closing the valves (V). The gases pass directly to the patient unless they are by passed by a stopcock (B) into the ether bottle. In the latter case the tension of ether vapour delivered to the patient will increase as the plunger (C) is lowered since then the gases are brought increasingly near the surface of the liquid ether or are made to bubble through it.

A rebreathing bag in the circuit acts as a reservoir to allow the lungs to expand with ease during inspiration. For an adult a flow of gases of 5 litres a minute is necessary to replenish the breathing bag and to ensure that a high percentage of the exhaled gases passes out

which are held in position by the spring band The gauze must not be thick enough to interfere with free respiration

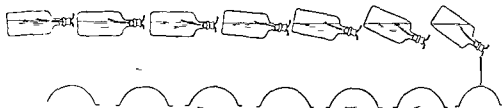


FIG 52 (After Waters)

Provided free regular respiration is not interrupted, the rate of flow of ether on to the mask must be continuous and must be increased uniformly

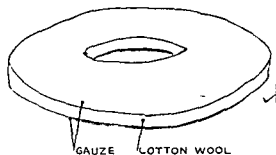


FIG 53

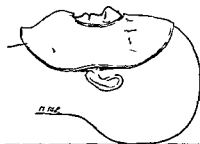


FIG 54



FIG 55

A gamgee pad—i.e. cotton wool covered with gauze—is a help to the efficient administration of ether by the open drop method

The concentration of ether vapour can be increased by inserting a gamgee pad underneath the mask thus preventing the patient from breathing any air which does not pass through the ether laden mask

valves to direct the flow of gases is unnecessary. In this 'to and fro' method the apparatus is simple, resistance to respiration is minimal, but airtight retention of the mask on the face is difficult on account of the position of the canister.

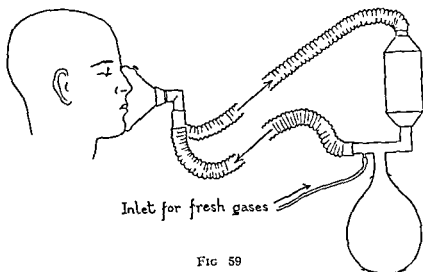


FIG 59

In the 'circle' method the introduction of valves allows the canister to be placed remotely. This makes control of the face-mask more easy. Compared with the 'to and fro' the circle apparatus is more complicated, and more resistance to respiration is offered by the valves and the greater length of breathing tubing necessary.

The advantages claimed for the  $\text{CO}_2$  absorption method of anaesthesia are (i) Maintenance of body heat. The anaesthetic gases are soon raised to body temperature and saturated with water vapour, and the patient continues to breathe this warm moist atmosphere. (ii) Diminished risk of explosion. Although elimination of this danger is not absolute the possibility of explosion is obviously reduced if inflammable gases are confined to a closed circuit. (iii) Vitiation of theatre atmosphere is prevented. With open drop ether the anaesthetist and operating team often work in an atmosphere heavily charged with ether vapour. (iv) Economy of anaesthetic gases is effected, and as a corollary the use of an expensive gas such as cyclopropane is made possible.

Read also

Waters R M 1936 *Carbon Dioxide Absorption Technic in Anesthesia*  
*Ann Surg* 103 38

through the expiratory valve near the face-piece. Such a flow limits the rebreathing into the corrugated tube and bag to a small volume, and the accumulation of  $\text{CO}_2$  is thus prevented.

It is worth recording that where ether vapour is carried to the patient by nitrous oxide and oxygen the resulting anæsthesia is frequently due almost entirely to the ether. The  $\text{N}_2\text{O}$  then can be considered merely as a vehicle and could be replaced by nitrogen without any noticeable difference in the anæsthesia.

### Closed circuit Method

Inhalation anæsthetics are excreted unchanged through the lungs. If a patient breathes in and out of a bag to which ether or any other inhalation anæsthetic is added until the desired level of anæsthesia is reached, theoretically this level could then be maintained indefinitely without further addition of anæsthetic. For life to be supported provision would have to be made for the supply of essential oxygen and for the removal of  $\text{CO}_2$  which if allowed to accumulate would soon reach a lethal concentration.

The mask is applied to the face firmly enough to prevent leakage.

The lungs and rebreathing bag then form a continuous closed circuit to which oxygen measured by a flowmeter, is added at the rate at which it is consumed by the body tissues. In the average adult at rest, this is about 250 c.c. per minute. A canister of soda lime is introduced into the circuit to absorb the  $\text{CO}_2$  expired. Ether (or other inhalation anæsthetic) is added gradually into the circuit until the requisite degree of anæsthesia is reached. Although in theory no more anæsthetic is now needed in practice it is found necessary to add small quantities from time to time in order to maintain a steady level

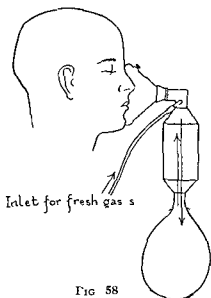


FIG 58

of anæsthesia. The additional anæsthetic is needed to maintain a constant concentration in the brain while tissue equilibrium is being established to compensate for small leakages and to replace small losses from the skin, wound surfaces and possibly by renal excretion.

If the soda lime canister is attached to the mask (fig 58) the use of

in general surgery is now rare and its use in dentistry is unwarranted. Its physical properties give chloroform a sphere of usefulness for emergency operations conducted away from hospital

### Dangers of Chloroform Anaesthesia

There are four well recognised causes of death from chloroform

1 Writers during the last century describe the occurrence of sudden death from psychological causes during induction of anaesthesia or early in operation. Half the deaths from chloroform have occurred within fifteen minutes of the beginning of induction. Chloroform sensitises the organism to adrenaline a drug which can produce ventricular fibrillation in man and animals. The result is that under chloroform anaesthesia a smaller dose of adrenaline than would otherwise produce fibrillation may now do so. There is a strong clinical impression that suppressed fear of anaesthesia or operation leads to liberation of the patient's own adrenaline into his blood stream in quantities sufficient if he is a susceptible subject to produce ventricular fibrillation in a heart sensitised by chloroform. Deaths from this cause will occur independently of the skill and experience of the anaesthetist. These sudden deaths occur notoriously in the healthy nervous patient who is dreading operation. In this connection it should be remembered that fear immediately before operation can now be overcome by the use of basal anaesthetics of which avertin, nembutal and thiopentone are familiar examples. Basal anaesthesia therefore may tend to protect the patient against this cause of sudden death from chloroform.

2 Death may occur from the direct action of strong chloroform vapour on the cardiac musculature of susceptible patients. Chloroform vapour in the inhaled mixture should not exceed 4 per cent.

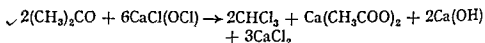
3 Death may result from paralysis of the respiratory centre from overdosage. This is heralded by increasing depression of respiration, and can be avoided by paying attention to respiratory signs. Should respiratory arrest occur, artificial respiration must be instituted at once but the probability of recovery is not as high as when a less toxic anaesthetic is used.

4 Death may arise also from delayed chloroform poisoning in which condition liver function becomes grossly disorganised. This grave complication the signs of which do not occur for some thirty six hours after administration, is more likely to occur in exhausted patients in those depleted of tissue fluids and in those who have been deprived of food. The risk of this complication increases in proportion to the degree of debility of the patient and to the amount of chloroform given.

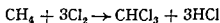
## CHAPTER XX

### CHLOROFORM

CHLOROFORM ( $\text{CHCl}_3$ ) was prepared independently by Guthrie, Liebig and Soubeiran in 1831. It is commonly manufactured by the reaction between acetone or alcohol and bleaching powder  $\text{CaCl}(\text{OCl})$ . This reaction is complex but omitting the intermediary stages the final result may be expressed as follows



It can also be prepared by the direct chlorination of methane gas ( $\text{CH}_4$ ). The reaction takes place in the presence of catalysts at temperatures above  $350^\circ \text{C}$



Other chlorination products are also formed and the chloroform is isolated by distillation. Chloroform is bottled in amber coloured bottles to exclude light, and as another precaution against the formation of phosgene ( $\text{COCl}_2$ ) 0.5 per cent alcohol is added

Chloroform is a colourless liquid with a sweet burning taste and an agreeable smell. It is sufficiently irritating to cause a mild burn if the lint on which it is dropped is kept in contact with the skin for some time. The specific gravity of the liquid is 1.5 and its boiling point  $61^\circ \text{C}$ . Chloroform vapour has a density of 4 and shares with nitrous oxide the great advantage of non inflammability.

Chloroform is highly toxic, anæsthesia being achieved by a poisoning of tissue cells appreciably greater than that produced by other anæsthetics. An even more serious deterrent to its use, however, is that during induction sudden death, which is unpredictable and apparently unavoidable occasionally occurs. It has the advantage over ether that its vapour is quite pleasant to inhale and on this account it was formerly in common use to produce unconsciousness before continuing anæsthesia with ether. This justification for its use has been eliminated since the introduction of basal anæsthetics. Moreover basal anæsthetics followed by  $\text{N}_2\text{O}$  and intravenous anæsthetics have replaced chloroform in most cases where anæsthesia has to be produced under conditions in which there is a danger of explosion. Its employment

about  $1\frac{1}{2}$ -2 ozs will be needed for the first hour of anæsthesia for a surgical operation on an average patient

In order to avoid too strong a concentration of chloroform vapour, Lister<sup>1</sup> in 1882 recommended that the mask should consist of the corner of a thin hand towel drawn through a closed safety-pin (fig 60). The towel should not touch the face, and although if necessary it may be kept moist with chloroform, the amount on it should never be sufficient to cause the chloroform to drop off the edges

## REFERENCE

- (1) Lister Joseph 1st Baron 1909 *Collected Papers* Oxford 1, 168



### Administration of Chloroform

Chloroform may be administered from a drop bottle on to a Schimmelbusch's mask covered with eight layers of gauze or one layer of flannel. A few drops are placed on the flannel and the mask gradually lowered on to the face. The rate of administration is increased as rapidly as the patient will tolerate. Swallowing, breath holding, or coughing are signs that the concentration of the vapour is being increased too quickly. If they occur the mask must be withdrawn a few inches, and gradually lowered as normal respiration is resumed. This precaution is necessary lest a period of breath holding be followed by an abnormally deep inspiration which might be charged

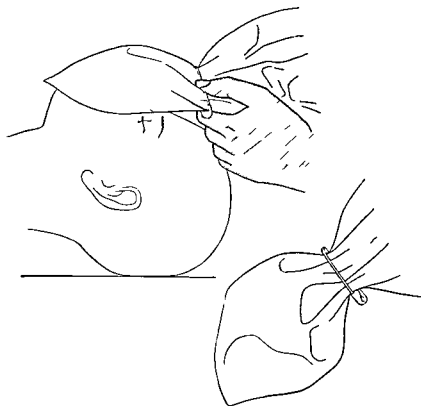


FIG 60

with a dangerously high percentage of chloroform vapour. The use of a gamgee pad over or under the Schimmelbusch's mask is never justified since this too may lead to a dangerous concentration of vapour. The amount of chloroform used for induction and maintenance of anæsthesia is strikingly less than in the case of ether. As a very rough guide

about 1½-2 ozs will be needed for the first hour of anæsthesia for a surgical operation on an average patient

In order to avoid too strong a concentration of chloroform vapour, Lister<sup>1</sup> in 1882 recommended that the mask should consist of the corner of a thin hand towel drawn through a closed safety-pin (fig 60). The towel should not touch the face, and although if necessary it may be kept moist with chloroform, the amount on it should never be sufficient to cause the chloroform to drop off the edges

#### REFERENCE

- (1) Lister Joseph 1st Baron 1909 *Collected Papers* Oxford 1, 168

### Administration of Chloroform

Chloroform may be administered from a drop bottle on to a Schimmelbusch's mask covered with eight layers of gauze or one layer of flannel. A few drops are placed on the flannel and the mask gradually lowered on to the face. The rate of administration is increased as rapidly as the patient will tolerate. Swallowing, breath holding, or coughing are signs that the concentration of the vapour is being increased too quickly. If they occur the mask must be withdrawn a few inches, and gradually lowered as normal respiration is resumed. This precaution is necessary lest a period of breath holding be followed by an abnormally deep inspiration which might be charged



FIG 60

with a dangerously high percentage of chloroform vapour. The use of a gamgee pad over or under the Schimmelbusch's mask is never justified since this too may lead to a dangerous concentration of vapour. The amount of chloroform used for induction and maintenance of anæsthesia is strikingly less than in the case of ether. As a very rough guide

recovery after short anaesthesia is quick Respiration throughout is shallow and quiet The gas is not irritant to the mucous membranes of the air passages or to the pulmonary epithelium, and therefore is suitable for patients with pulmonary disease

### Administration of Cyclopropane

Before the mask is applied the rebreathing bag should be filled with sufficient oxygen to allow comfortable respiration The flow of oxygen is then adjusted to satisfy the patient's metabolic requirements (i.e. about 250 c.c. per minute for an adult) and the cyclopropane is introduced into the circuit at a rate of 500-1 000 c.c. per minute This flow is kept up until the desired anaesthesia is achieved the level of which is then maintained by supplying extra cyclopropane at intervals or as a continuous trickle at the rate of about 50 c.c. per minute The reason why this additional anaesthetic is required is explained on p 178

The average percentages of cyclopropane in the inhaled mixture which would produce unconsciousness light anaesthesia, moderately deep surgical anaesthesia and respiratory arrest respectively, are 5, 10, 20, and 40, but such figures must be taken merely as helpful indications of what may be expected in the average patient for as with all narcotics the administrator must be guided by the reactions of the individual patient and not by rule-of thumb

### REFERENCES

- (1) Lucas G H W and Henderson V E 1929 *Canad med Ass J*, 21, 173-5

Read also

Robbins B H 1940 *Cyclopropane Anesthesia* Baltimore

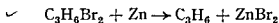
Waters R M 1936 *Brit med J* 2, 1013

Waters R M and Schmidt E R 1934 *J Amer med Ass* 103, 975

## CHAPTER XXI

### CYCLOPROPANE

CYCLOPROPANE ( $C_3H_6$ ) was first prepared by Freund in 1882 by treating dibromopropane with zinc at high temperatures in the presence of a catalyst



Dibromopropane is now obtained from natural gas containing a high proportion of propane ( $C_3H_8$ ) by treatment with bromine

After preliminary tests in the laboratory by Lucas and Henderson in 1929<sup>1</sup> cyclopropane was introduced into clinical anaesthesia by Waters after he himself had been anaesthetised with it by his then First Assistant E A Rovenstine

Cyclopropane is a colourless gas with a not unpleasant naphtha like smell. It is one and a half times as heavy as air and widely varied mixtures with oxygen or air are highly explosive. It liquefies at a pressure of 75 lb. per square inch and is supplied as a liquid in cylinders. Its price per gallon is approximately fifty times that of nitrous oxide but since it is always administered in a closed circuit apparatus (p 178) the cost of cyclopropane anaesthesia is approximately the same as that of other inhalation anaesthetics

With this potent agent, anaesthesia suitable for any surgical procedure can be obtained but because of the difficulty in operations on the head and neck of establishing a gastight connection between apparatus and patient without encroaching on the field of operation it is seldom used in dentistry

Cyclopropane is not suited for use by the occasional anaesthetist. The signs of anaesthesia correspond closely to those of ether, but the successive stages are passed through with much greater rapidity. Muscular relaxation cannot be relied upon when the drug is used in low concentrations and an attempt to produce relaxation by increasing the cyclopropane content of the mixture may be accompanied by marked respiratory depression and cardiac irregularities. Nevertheless in the hands of the experienced anaesthetist it furnishes eminently satisfactory results and by many is considered to be the anaesthetic of choice for most surgical operations

Induction with cyclopropane is rapid and not unpleasant and



## CHAPTER XXII

## CURARE AND OTHER MUSCLE RELAXANTS

CURARE which was introduced into anæsthesia only a few years ago but which has already proved itself an important advance is a substance which from ancient times has enjoyed a respected reputation among the South American Indians. It is the basis of their famous arrow poison or flying death tales of which have been told in the last few hundred years by travellers returning from the Amazon. Some of these travellers in particular Charles Waterton<sup>1</sup> an Englishman who explored South America in 1812 brought back accurate accounts of the way in which death was produced by poisoned arrows. Secrecy has always surrounded the details of preparation of native specimens and the medicine man who could produce a potent sample was an important individual. Different specimens from native sources vary in their action and only in recent years have pure preparations become available.

H. King<sup>2</sup> in 1935 first obtained d tubocurarine chloride in its pure state. An American R. C. Gill<sup>3</sup> brought supplies of crude curare to the United States in 1938 and the active principle was extracted by the house of Squibb. In 1940 Dr L. Wright of Squibb's suggested to Griffith the anæsthetist of Montreal that curare might be used successfully in anæsthesia to produce muscular relaxation. Griffith was understandably hesitant about following up this novel idea but he finally gave it a trial in 1942<sup>4</sup>.

### THE PHARMACOLOGY AND PHYSIOLOGY OF D-TUBOCURARINE AND ITS ANTIDOTE, PROSTIGMIN\*

Curare is a crude plant extract obtainable from several species of tropical plants. Different species vary greatly in composition, and the crude extract is quite unfit for clinical use. It contains many alkaloids some of which paralyse skeletal muscle by producing neuro muscular block, some have a central convulsant action and others relax spastic muscle. The word curare has thus too wide a meaning to be correctly used to denote a preparation which produces neuromuscular

\* I am greatly indebted to W. Feldberg M.D. F.R.S. for advice and assistance with this section. F. B. B.

block. The only alkaloid of clinical interest obtained from preparations of curare is *d* tubocurarine and this is sometimes incorrectly referred to as 'curarine,' which is another different and definite curare alkaloid. In clinical doses *d* tubocurarine has scarcely any action other than that of paralysing skeletal muscle by blocking conduction at the neuromuscular junction.

Our ignorance of the exact nature of the crude extracts of curare is revealed by the fact that these are still referred to by the names of the containers in which they are delivered to the importers. This curious distinction is not quite so illogical as at first appears, since their properties differ and they are, therefore different in botanical as well as in geographical origin. The three types are *tube curare* derived from French Guiana where the crude extract is filled into the hollows of bamboo tubes, *calabash or gourd curare*, from British Guiana and the Upper Amazon, where the extract is put into calabashes or gourds, and *pot curare*, from the Upper Orinoco, where a pot is used as container. The alkaloids derived from these sources are as follows. *tube curare* yields *tubocurarine*, which produces neuromuscular block and *curine*, which is weaker in action and toxic to the heart. *Calabash curare* yields *curarine* which is extremely potent and has some action similar to that of tubocurarine but has also a central convulsant action similar to that of strychnine. *Pot curare* yields *protocurine*, *protocuridine*, and *protocurarine*, the latter being the only one with a curariform action.

*d* tubocurarine chloride was first obtained in the pure state from tube curare in 1935 by H. King<sup>2</sup> who worked out its chemical structure. It belongs to a group of compounds known as *quaternary ammonium salts* which in general have the property of paralysing neuromuscular conduction at the neuromuscular junction. King suggested that plants of the *Chondrodendron* species were those from which the active principle was derived although in tube curare these are mixed with other plants and with gums and resins. Wintersteiner and Dutcher<sup>3</sup> of the Squibb Institute who followed up this suggestion isolated *d* tubocurarine as the active principle of *Chondrodendron tomentosum*. Thus from an astonishing mixture of varieties of curare derived from unknown botanical sources it has now been possible to prepare one of the active toxic principles from a known source. Squibb<sup>3</sup> prepared from this source a purified curare extract of standard potency which they named *Intocostrin*. This is stated to contain 3 mg. of tubocurarine per c.c. and negligible quantities of other curare alkaloids. It owes its curariform action almost entirely to the *d* tubocurarine chloride present. Squibb's Intocostrin was the first prepara-



## CHAPTER XXII

## CURARE AND OTHER MUSCLE RELAXANTS

CURARE which was introduced into anæsthesia only a few years ago but which has already proved itself an important advance is a substance which from ancient times has enjoyed a respected reputation among the South American Indians. It is the basis of their famous arrow poison or flying death tales of which have been told in the last few hundred years by travellers returning from the Amazon. Some of these travellers in particular Charles Waterton<sup>1</sup> an Englishman who explored South America in 1812 brought back accurate accounts of the way in which death was produced by poisoned arrows. Secrecy has always surrounded the details of preparation of native specimens and the medicine man who could produce a potent sample was an important individual. Different specimens from native sources vary in their action and only in recent years have pure preparations become available.

H. King<sup>2</sup> in 1935, first obtained d tubocurarine chloride in its pure state. An American R. C. Gill<sup>3</sup> brought supplies of crude curare to the United States in 1938 and the active principle was extracted by the house of Squibb. In 1940 Dr L. Wright of Squibb's suggested to Griffith the anæsthetist of Montreal that curare might be used successfully in anæsthesia to produce muscular relaxation. Griffith was understandably hesitant about following up this novel idea but he finally gave it a trial in 1942<sup>4</sup>.

### THE PHARMACOLOGY AND PHYSIOLOGY OF D-TUBOCURARINE AND ITS ANTIDOTE, PROSTIGMIN\*

Curare is a crude plant extract obtainable from several species of tropical plants. Different species vary greatly in composition, and the crude extract is quite unfit for clinical use. It contains many alkaloids some of which paralyse skeletal muscle by producing neuro muscular block, some have a central convulsant action and others relax spastic muscle. The word curare has thus too wide a meaning to be correctly used to denote a preparation which produces neuromuscular

\* I am greatly indebted to W. Feldberg M.D. F.R.S. for advice and assistance with this section. F. B. B.

unconsciousness. The paralysing effect of *d* tubocurarine chloride on skeletal muscle is that which interests us most from the point of view of anaesthesia. In man the skeletal muscles are affected by curarisation in the following order—the muscles supplied by the cranial nerves (face and neck) are affected first then those of the limbs, back, abdomen, intercostals, and finally the diaphragm. Recovery of muscle function occurs in the reverse order.

In order to understand how *d* tubocurarine chloride (and other neuromuscular blocking agents) act it is necessary to review the mechanisms concerned in contraction of skeletal muscle. The nerve to skeletal muscle ends on a specialised structure of the muscle fibre, the motor endplate. It is now generally assumed that acetylcholine is released by the arrival of the nerve impulse at the nerve ending and that the acetylcholine then acts on the motor endplate. Its action consists of a depolarisation of the endplate which can be recorded and which is called the motor endplate potential. This localised depolarisation excites the adjacent parts of the muscle fibre so that the propagated excitation wave, the action potential, passes over the muscle fibre and this excitation wave in its turn leads to contraction of the contractile elements of the fibre.

Under normal physiological conditions the acetylcholine is quickly hydrolysed by the true cholinesterase present at the motor endplate region. Therefore the endplate is repolarised before the muscle fibre has come out of its refractory state so that a single nerve impulse gives rise to a single wave of excitation and of contraction only.

What happens when acetylcholine persists at the endplate region must now be discussed. Two different effects may occur. When the depolarisation outlasts the refractory period of the muscle fibre it becomes re-excited and this may occur several times. In that case the single nerve impulse gives rise to several waves of excitation resulting in a summated contraction. The beneficial effect of prostigmin (p. 197) as an antidote to *d* tubocurarine chloride and for the treatment of myasthenia gravis is explained on these lines. The second change which may occur is more profound and leads to neuromuscular block. It occurs particularly when acetylcholine persists in excess at the endplate region. This effect can be imitated by other depolarising substances. It is explained as follows: the depolarisation spreads to adjacent regions of the muscle fibre on either side of the endplate; it is not the acetylcholine itself which spreads but the depolarisation current, as recently shown by Burns and Paton<sup>11</sup>. If a part of a muscle fibre however becomes depolarised it becomes electrically inexcitable and prevents the passage of an

tion of curare to be used in anæsthesia Griffith, of Montreal, who first employed it in 1942<sup>4</sup> had used it in about 300 cases only until 1945<sup>6,7,8</sup> but since that time its use has rapidly increased J Halton<sup>9</sup> in England first used *d* tubocurarine chloride (Tubarine Burroughs Wellcome and Co) in 1944 in anæsthesia for thoracic surgery, and since then supplies have become much more readily available and *d* tubocurarine chloride is now a frequent adjunct to anæsthesia In England Burroughs Wellcome and Co in 1936 made available a solution of *d* tubocurarine chloride, prepared from crude curare obtained from the South American *Chondrodendron tomentosum* This is now sold under the trade name of Tubarine and is a stabilised solution containing 10 mg per c c of *d* tubocurarine chloride

Intocostin and Tubarine are not miscible with solutions of thiopentone with which they form a deposit and must therefore be given in separate injections Now however Burroughs Wellcome and Co have prepared Tubarine Miscible, a pure solution of *d* tubocurarine chloride in saline When Tubarine Miscible is added to a 5 per cent thiopentone solution, a precipitate of thiopentone acid is formed immediately, and this re dissolves in the thiopentone (which is alkaline) on shaking In mixing solutions of Tubarine Miscible and thiopentone there should be at least 1 c.c. of 5 per cent, thiopentone solution for every 1 mg. of *d* tubocurarine chloride (0.1 c.c. Tubarine Miscible)

*d* tubocurarine chloride is effective only if injected intravenously or subcutaneously and not at all if taken by mouth which explains the safety of the native who ate the animals he killed by poisoned arrows This ineffectiveness is not due to destruction of the drug by the digestive juices but to its detoxication by the liver *d* tubocurarine chloride is rapidly metabolised after intravenous administration Some of it is excreted by the kidneys either unchanged or in a form still capable of curarising and some is detoxified by the liver

As long ago as 1850 Claude Bernard<sup>10</sup> published his classical account of experiments performed in the previous years which showed that extracts of curare prevented the passage of an impulse from nerve to voluntary muscle by interference with transmission at the neuromuscular junction Since that time curare preparations of varying potency and activity have been used by physiologists for this purpose These extracts containing an unknown mixture of curare alkaloids had an effect on both the neuromuscular mechanism and on the nervous system some of the alkaloids present (especially curarine) having a convulsant action Pure *d* tubocurarine chloride acts almost entirely on the neuromuscular junction and has no significant central stimulant depressant or analgesic action in man It does not cause

prostigmin symptoms. One of these drugs must therefore always be given when prostigmin is used clinically.

[The effects of acetylcholine on the autonomic ganglia and on the motor endplates of skeletal muscle are called the nicotine like effects, they are resistant to atropine. It is interesting to note that prostigmin is the drug used for the treatment of myasthenia gravis, a disease characterised by weakness of skeletal muscle which is imitated by curarisation—in fact curarisation converts the subject into a temporary myasthenic, and d tubocurarine chloride should not be given to known myasthenics who are highly sensitive to it

The motor endplate of skeletal muscle and the autonomic ganglia have much in common in their response to certain drugs. For instance d tubocurarine chloride antagonises not only the depolarising action of acetylcholine on the motor endplate, but also the depolarising action of acetylcholine on the ganglion cells which is responsible for their excitation. The pronounced depression of laryngeal and bronchial reflexes which is of great importance to the anaesthetist may be due to this action of d tubocurarine chloride. Its effect on the intestine during anaesthesia is variable: some observers have noted contraction whilst others have observed diminished irritability. It must be remembered however that in clinical anaesthesia other drugs which also effect the autonomic ganglia, especially atropine or scopolamine are commonly employed for premedication and will be exerting their effect at the same time as d tubocurarine chloride thus modifying the effect of that drug on the autonomic system. In fact, one of these drugs should always be given as premedication when d tubocurarine chloride is to be used since the latter causes salivation.

Bronchospasm resulting from the administration of curare compounds has been described by various writers. West<sup>12</sup> using a crude curare extract noted its occurrence in dogs and Cole<sup>13</sup> found difficulty in inflating dogs immediately after giving large doses of Intocostin. This may be due to a histamine effect on the bronchi as histamine like wheals are produced by intracutaneous injection of d tubocurarine chloride in man and histamine is released from the tissues by the injection of d tubocurarine chloride into animals. Prescott<sup>14</sup> believes that there may be some risk of bronchospasm in man if the patient is lightly anaesthetised or conscious and if the dose of curarising drug is on the low side as he has observed it in four cases: one a conscious volunteer and three mental patients undergoing electrical convulsion therapy and very lightly narcotised with thiopentone. He recommends that it should be treated by the administration of thiopentone or of more d tubocurarine chloride.

excitation wave. Therefore such a depolarised region must act as a block for the nerve impulse.

There are two entirely different mechanisms by which neuromuscular block may be established—either by reducing the depolarising effect of the released acetylcholine or by producing excessive depolarisation itself. *d* tubocurarine chloride acts in the former and decamethonium (C 10) in the latter way.

*d* tubocurarine chloride produces neuromuscular block by antagonising the depolarising action of acetylcholine on the motor endplate. The acetylcholine released from the motor nerve ending is no longer able to depolarise the endplate sufficiently to excite the muscle fibre but *d* tubocurarine does not interfere with the release of acetylcholine by the nerve impulse. During curarisation larger amounts of acetylcholine are required than normally to produce effective depolarisation of the endplate and thus to establish neuromuscular transmission. This brings us to the discussion of the physiological antidote to *d* tubocurarine chloride which is physostigmine, an alkaloid derived from the calabar bean of New Guinea.

The preparation used clinically as an antidote to *d* tubocurarine chloride is prostigmin, a synthetic preparation with a similar action to that of the alkaloid physostigmine. The action of prostigmin is to inactivate the true cholinesterase, the enzyme which hydrolyses acetylcholine and which is present in high concentration at the neuromuscular junction so that it no longer destroys acetylcholine. When prostigmin is given, acetylcholine is thus able to accumulate at the motor endplate and in a curarised subject this excess of acetylcholine overcomes the effect of *d* tubocurarine chloride so that the endplate becomes sufficiently depolarised to excite the muscle fibre again.

The effects of prostigmin are not confined to the neuromuscular junction. Acetylcholine is released not only at the motor nerve endings but also at the endings of the parasympathetic and of some sympathetic fibres (those to the sweat glands in man), and at the pre-ganglionic endings at sympathetic and parasympathetic ganglia. If prostigmin is given it will inactivate the cholinesterase at all these different sites and the effects of the accumulated acetylcholine released from parasympathetic nerve endings are clinically particularly striking. These parasympatho mimetic effects of acetylcholine and of prostigmin are termed muscarine like actions, and consist of smooth muscle contractions, secretions of glands and cardiac inhibition. They are annulled by atropine or by scopolamine which apart from its central action has a peripheral atropine like action and theoretically it should therefore protect as well as atropine against muscarine like

so that artificial respiration was necessary to prevent asphyxia. There was no analgesic action whatsoever. Tractional administration was tested to see if it would produce relaxation without respiratory suppression but was found not to do so since 30 mg given intravenously at the rate of 1 mg per minute did not produce curarisation and the subject could sit up 10 minutes after the injection was completed. A curarising dose in the conscious patient was found to last for 20 to 30 minutes.

The value of curarisation in anaesthesia is that it enables the anaesthetist to produce extreme muscular relaxation without administering a large dose of general anaesthetic. The benefit to the patient is a more swift and pleasant recovery. Since *d* tubocurarine chloride does not itself produce analgesia and since its use prevents recognition of the degree of general anaesthesia used concomitantly (the signs of depth of anaesthesia being muscular signs) (p. 67) it is essential for the anaesthetist to be certain that general anaesthesia is adequate to produce and maintain unconsciousness. Inadequate depth of anaesthesia is sometimes suggested by a rising pulse-rate but other causes may be responsible for this sign which cannot therefore be relied upon. If maintenance of general anaesthesia is not ensured the patient may be completely aware of all that is going on and may experience the full pain of the operation without being able in any way to make a protest. This possibility must always be borne in mind and only his experience of general anaesthesia will enable the anaesthetist to avoid it.

Prescott Organe and Rowbotham<sup>17</sup> state that there is an optimal dose of *d* tubocurarine chloride less than which will not produce adequate relaxation whilst very little more produces diaphragmatic paralysis. If this occurs full respiratory exchange must be maintained by inflation of the lungs until normal respiratory movements return. In practice it is found that in fit adults the optimal dose is 15 to 20 mg. This produces abdominal relaxation which lasts for 30 to 45 minutes. The degree and duration of relaxation depend also on the amount of general anaesthetic given at the same time.

The limits of the optimal dose are narrow especially in children. For the very young, the aged, and in cachectic and severely ill patients the dose of *d* tubocurarine chloride should be reduced.

It is best to give the initial curarising dose estimated to be needed all at once. To give *d* tubocurarine chloride in divided doses leads only to a larger total dose being given and to delay in development of a satisfactory degree of abdominal relaxation. For operations continuing longer than about 45 minutes after which time the effect of the initial dose will be wearing off relaxation can be maintained by

When using pure *d* tubocurarine chloride it is however, a condition which is met extremely rarely in clinical anæsthesia and I have in fact seen only one case out of hundreds in which *d* tubocurarine chloride was used where inflation of the lungs was difficult after curarisation. This was in a frail patient for gastrectomy and the condition was much relieved by the administration of more thiopentone and though there remained slight resistance to inflation for some 30 minutes there was no difficulty in keeping the patient well-oxygenated. It appears that general anæsthesia protects against the histamine effect of *d* tubocurarine chloride

Some observers<sup>15 16</sup> believe that ether has a curariform effect and that when it is used the dose of *d* tubocurarine chloride should be reduced. I do not consider that the evidence for this is conclusive and it is in any case difficult to assess how much relaxation produced is due to the general anæsthetic given and how much to the *d* tubocurarine chloride. The only guide to procedure is the anæsthetist's experience and I have found no reason to reduce the dosage of *d* tubocurarine chloride when using ether as the general anæsthetic since in such cases I produce only light anæsthesia with ether.

*d* tubocurarine chloride in clinical doses has no effect on the electrocardiogram in man and little or no effect on the blood pressure<sup>9 14</sup>. There is as yet no evidence that it can cause death other than by asphyxia from unrelieved diaphragmatic paralysis and therefore, the most important rule for the anæsthetist using a curarising drug is to maintain full respiratory exchange at all times. No one who is not competently and confidently capable of dealing with the apnoic patient should venture to use curarisation as an aid to relaxation in anæsthesia.

### THE USE OF D-TUBOCURARINE CHLORIDE IN ANÆSTHESIA

*d* tubocurarine chloride given intravenously to the unanæsthetised subject produces progressive muscular paralysis without loss of consciousness. Experiments by Prescott Organe and Rowbotham<sup>17</sup> (the first named being the experimental subject) showed that in a fit adult 10 mg of *d* tubocurarine chloride had very little effect on voluntary muscle. 20 mg produced paresis of the muscles of the face, neck, arms, legs and abdomen in that order with loss of speech but without loss of the cough reflex and although respiration was not consciously affected, spirometer readings showed that the tidal air was reduced by about one third. 30 mg produced complete paresis of all voluntary muscle and almost complete cessation of respiration.

lungs because of the relaxation of oesophageal muscles and of the cardiac sphincter of the stomach. This condition hampers the surgeon during abdominal operations and should be relieved by passage of a wide bore stomach tube. Further after any operation for which *d* tubocurarine chloride has been used the epigastrium should be examined before the patient leaves the operating theatre and if the stomach is found to be distended a stomach tube should be passed. If the anæsthetic is given through a wide bore endotracheal tube connected directly to the anæsthetic machine gases will pass into the lungs alone during inflation and none will enter the stomach.

The anæsthetist using *d* tubocurarine chloride must be prepared to deal with an apnoic patient and no one who is not confident of his ability to cope with apnoea should venture to use this drug. It is necessary to be extremely watchful for any degree of respiratory depression whatsoever. Respiration may often need to be assisted in order to maintain full respiratory exchange—it is not enough to be satisfied with the continuance of respiratory movements for these are usually incomplete for a short time immediately after a curarising drug has been given in full dosage. A brief period of complete diaphragmatic paralysis often occurs during which it is essential to employ artificial or controlled respiration. If these points are watched with extreme care then *d* tubocurarine chloride can be used with safety for probably the only way in which it can cause death is by asphyxia.

### Recovery from Anæsthesia with Curarisation

When *d* tubocurarine chloride has been used recovery from anæsthesia is much more rapid than after general anæsthesia used alone for any except minor procedures. There are no undesirable after-effects resulting from the *d* tubocurarine chloride itself and as a rule the amount of general anæsthetic given has been so small that this is rapidly eliminated. The patient's swift recovery minimises the length of time for which individual nursing care is required in the immediate post-operative period and it is well to remember that post-operative sedatives will have to be given soon after the patient returns to the ward.

### Indications for the Use of *d* tubocurarine Chloride

*d* tubocurarine is used chiefly in abdominal surgery. Here it provides excellent operating conditions comparable with those obtained



giving more *d* tubocurarine chloride, but the second and any subsequent doses must be much smaller than the initial dose e.g. 5 mg or even 2.5 mg intravenously is usually a satisfactory second dose when the first has been 20 mg and this further dose will produce approximately another half hour's relaxation

### Technique of Administration

Anæsthesia is induced with thiopentone and is stabilised in the first plane with any chosen inhalation anæsthetic (nitrous oxide oxygen ether or cyclopropane) The full curarising dose of *d* tubocurarine chloride is then given intravenously i.e. 15 to 20 mg for the normal adult Full muscular relaxation develops in 2 to 3 minutes and general anæsthesia is then maintained by the chosen method. One method used by R. R. Macintosh because of the previous knowledge gained from experience with uncurarised patients that the percentage of ether given will maintain unconsciousness, is the administration of ether vapour 5 per cent by means of the Oxford Vaporiser

If 5 minutes after the *d* tubocurarine chloride has been given the surgeon reports that relaxation is not adequate then more must be administered to produce the required effect. This dose should be 5 to 10 mg and not a full curarising dose. As a rule all the relaxation required will by now have been produced and will last for 30 to 45 minutes after which time under light general anæsthesia there will be signs of returning muscular tone. If the operation is not completed the decision must then be made whether to develop relaxation again by the administration of more *d* tubocurarine chloride (e.g. 2.5 to 5 mg) or whether to increase the depth of general anæsthesia. If the operation is expected to finish soon the latter is the more satisfactory method as it avoids the risk of having the patient deeply curarised and with respiration depressed by the time he is due to return to the ward.

To cachectic, frail and elderly patients a smaller initial dose of *d* tubocurarine chloride should be given e.g. 5 to 10 mg at the start, until its effect is seen this dose being added to as required Children also require smaller dosage. Up to 5 mg intravenously for children up to the age of twelve is usually a sufficient curarising dose.

Intubation of the larynx though not essential when *d* tubocurarine chloride is used, is frequently performed in order to ensure perfect control of the airway and to facilitate inflation of the lungs It is advisable when the Trendelenburg position is to be used

It should be remembered when using *d* tubocurarine chloride that it is possible to distend the stomach with gases when inflating the

it causes another muscle relaxant myanesin which produces less depression has gained greater popularity for use against this disease (p 201)

Outside the realm of anaesthesia *d* tubocurarine chloride has also a use in electrical convulsion therapy<sup>20</sup> to soften or cushion the effects of the electric shock. This is a great advantage and enables the injuries previously encountered, such as fractures, to be avoided in a patient undergoing convulsion therapy. It also enables patients such as hypertensive, arteriosclerotic and senile patients in whom unmodified electrical convulsion therapy would be dangerous if not absolutely contra indicated to be treated with safety. For this purpose however, *d* tubocurarine chloride has been superseded by C 10 (p 199)

### The Antidote to *d*-tubocurarine Chloride

The physiological antidote to *d* tubocurarine chloride is prostigmin as has already been explained. Prostigmin is given intravenously and prior to its injection atropine or scopolamine either of which annul its muscarine like effects such as cardiac inhibition and excessive salivation should always be given.

As a rule if spontaneous respiratory movements have begun, a dose of 2.5 mg. of prostigmin is sufficient to effect recovery of normal respiration. It is rarely necessary to exceed 5 mg and excessive dosage of prostigmin may produce toxic effects indistinguishable from the effects of *d* tubocurarine chloride itself. The mechanism by which this condition of paralysis is produced is at present unknown.

If atropine or scopolamine has been given within 2 hours of the time when it is decided to administer prostigmin, no more need be given but otherwise, gr  $\frac{1}{100}$  to gr  $\frac{1}{50}$  of atropine should be injected intravenously 5 minutes before the prostigmin since prostigmin acts more quickly than atropine. This procedure protects against the undesirable cardiac effects of prostigmin and is especially important in patients with any degree of cardiac failure—from pre existing cardiac disease shock or toxæmia—for in them the sudden gross bradycardia which prostigmin sometimes causes might have serious consequences.

Normally the response to prostigmin is dramatic and is seen within 2 minutes. Respiration will usually return to normal and muscular tone will at the same time be restored. If the effect of the general anaesthetic has worn off the patient will recover from muscular paralysis and it will be evident that he is conscious. This desirable effect is what is usually seen but prostigmin is not without its dangers

under spinal anæsthesia. In fact the use of *d* tubocurarine chloride which can be administered so quickly and easily by intravenous injection has practically ousted the previously popular intercostal block.

It is also used according to the anæsthetist's choice for a wide variety of other operations—e.g. for thoracic operations for bronchoscopy (where its depressant effect on the laryngeal reflex as well as its relaxant effect on skeletal muscle is especially useful), for tonsillectomy and for Cæsarean section as described by Gray.<sup>19</sup> In every case its use reduces the amount of general anæsthetic required. For Cæsarean section after premedication with gr  $\frac{1}{100}$  of atropine only anæsthesia is induced with 0.25 to 0.5 g of thiopentone and light general anæsthesia maintained with any chosen inhalation anæsthetic. 15 mg of *d* tubocurarine chloride is given intravenously as soon as possible after induction of general anæsthesia. Relaxation is excellent and the newborn babe usually cries lustily at once showing no evidence of curarisation. This clinical experience has been investigated by Buller and Young<sup>20</sup> whose experimental work shows that though the full term foetus of humans and animals is susceptible to the action of *d* tubocurarine chloride this drug does not cross the placental barrier in appreciable amounts and therefore does not affect the foetus *in utero*.

*d* tubocurarine chloride facilitates laryngeal intubation as it dampens or abolishes the laryngeal reflex and enables laryngoscopy and intubation to be performed at light levels of general anæsthesia.

There is one circumstance in which *d* tubocurarine chloride can be used with valuable therapeutic effect—in the relief of intense laryngeal spasm (p. 312). This is seen at its worst in sthenic individuals and where it occurs e.g. during induction of anæsthesia for an operation where it was not originally intended that *d* tubocurarine chloride should be used it can be relieved almost instantaneously by the injection of a curarising dose—as soon as the relaxant effect of the *d* tubocurarine chloride develops inflation of the lungs with oxygen (impossible during the period of intense laryngeal spasm) can proceed fully and without delay and with a gratifying return to the normal florid pink of the previously purple complexion.

### Other Uses of *d*-tubocurarine Chloride

*d* tubocurarine chloride has also been used in the treatment of tetanus a disease for which the services of the anæsthetist are increasingly requested. Because of the respiratory depression which

of the active drug. It is miscible with solutions of thiopentone

Flaxedil is a satisfactory relaxant, the dose equivalent to a 15 mg dose of *d* tubocurarine chloride being 80 mg.<sup>22</sup> Relaxation develops within 3 minutes and the effect of a curarising dose lasts for 25 to 30 minutes. Later doses if required to continue relaxation should be smaller than the initial dose and usually 40 mg is a satisfactory second dose.

Flaxedil does not depress respiration so much as a comparable dose of *d* tubocurarine chloride, but its effect is not so long-lasting and tachycardia occurs in about 40 per cent of cases. No ill effects have been reported as a result of this feature. The indications and contraindications for the use of flaxedil are the same as those for *d* tubocurarine chloride.

The antidote to flaxedil is prostigmin as with *d* tubocurarine chloride.

### C 10

Decamethonium iodide commonly known as C 10 is bis(trimethylammonium) decane didecylate a synthetic relaxant made by Allen and Hanburys Ltd. which acts on the neuromuscular junction. It is miscible with solutions of thiopentone. C 10 is a satisfactory relaxant similar in its effects to *d* tubocurarine chloride but producing these by a fundamentally different mechanism.<sup>21, 23</sup> C 10 causes a specific and abnormally persistent motor endplate depolarisation which spreads to the adjacent regions of the muscle fibre. These zones become electrically inexcitable and this results in neuromuscular block. The action is thus analogous to that produced by an excess of acetylcholine at the motor endplate. Prostigmin which is an effective antidote against relaxant drugs that produce neuromuscular block in the same way as *d* tubocurarine chloride because it inactivates cholinesterase would not be expected to be effective against C 10. In theory it would increase the degree of block which C 10 produces since it allows the released acetylcholine to accumulate thereby increasing the depolarisation. C 10 is the first neuromuscular blocking agent acting by a direct depolarising action to be used clinically. The effects of *d* tubocurarine chloride and of C 10 should be compared under similar conditions i.e. during anaesthesia for it to be possible to make a correct assessment of their behaviour as clinical relaxants. Experiments on conscious patients do not permit of a valid comparison for circulating adrenaline minimises the relaxant effect of C 10 and increases this action of *d* tubocurarine chloride. It is incorrect to refer to C 10 as a curarising drug since its effect is

and in one recorded instance—occurring it is true in a gravely ill patient—the injection of 2.5 mg of prostigmin with gr  $\frac{1}{100}$  of atropine was followed within 2 minutes not by the expected respiratory response but by cyanosis and cardiac arrest <sup>21</sup>

Prostigmin should be used when necessary but I do not agree with its routine administration to every patient who has received *d* tubocurarine chloride. Respiration must, of course be adequate before the patient leaves the operating theatre and if by misjudgment too much *d* tubocurarine chloride has been given or if the operation finishes more quickly than has been anticipated, the anæsthetist must either continue to assist respiration artificially until normal respiration returns or he must give prostigmin to achieve this result

### Contra-indications to the Use of *d* tubocurarine Chloride

*d* tubocurarine chloride should not be given to any patient known to have myasthenia gravis for it aggravates this condition and the usual dose for a normal individual is a gross overdose for a myasthenic

In cases of intestinal obstruction and lung abscess *d* tubocurarine chloride should not be used until the anæsthetist is convinced that the danger of inhalation of secretions is in the first case avoided (by stomach washout immediately prior to operation the stomach tube should be left in position during induction and operation) and in the second case minimised by preparations being ready for immediate bronchoscopy and suction drainage if necessary

Ⓒ Gross renal damage is a contra indication to the use of *d* tubocurarine chloride which is in part excreted by the kidneys

### OTHER MUSCLE RELAXANTS

Since the introduction of *d* tubocurarine chloride other muscle relaxants have been produced and doubtless many more will follow in due course. For the present for anæsthetic purposes *d* tubocurarine chloride is still the choice of the bulk of anæsthetists though the other relaxants each have their own claims to usefulness and their advocates

#### Flaxedil

Flaxedil is tri (diethylaminoethoxy) benzene triethyliodide or gallamine triethyliodide a synthetic relaxant made by May and Baker Ltd. It produces neuromuscular block in the same way as *d* tubocurarine chloride. It is supplied as a 4 per cent solution in ampoules of 2 c.c. and 3 c.c. these containing respectively 80 mg. and 120 mg.

already described. Whereas all the others produce their effect by causing neuromuscular block, myanesin acts by depressing parts of the nervous system between the cortex and the spinal cord.<sup>27</sup> It may in addition depress function in the basal ganglia, brain stem and thalamus as it is reported to relieve pain of thalamic origin and to reduce Parkinsonian tremor and rigidity.<sup>28</sup> It also arrests epileptic attacks.<sup>29</sup>

Myanesin was first introduced as a relaxant for use in anaesthesia but its depressant effect on the reflex excitability of the spinal cord makes it especially valuable in the treatment of tetanus. Myanesin does not paralyse muscle but markedly reduces its tone and thus without much depressant effect on respiration.<sup>30</sup> Mallinson<sup>31</sup> stated that the dose affecting the vital centres in animals was ten times that which caused muscular relaxation. In the doses required for the treatment of tetanus myanesin has no effect on respiration and is therefore superior for this purpose to *d* tubocurarine chloride which in effective doses may produce respiratory paralysis.

Myanesin is supplied in ampoules of 10 c.c. each containing 1 g. of the active drug. It may be given intravenously or intramuscularly and for the treatment of tetanus 1 g. for an adult is given as required to control the spasms. Myanesin is miscible with solutions of thiopentone.

Myanesin has two disadvantages which diminish its popularity as a relaxant for anaesthetic purposes. Intravenous injection of the 10 per cent. solution has led frequently to local venous thromboses and such an injection produces in many subjects intravascular hæmolysis.<sup>32</sup> The larger the dose the greater the likelihood and the degree of hæmolysis. Hæmoglobinuria does not always occur as the renal threshold for hæmoglobin is usually high. If myanesin is given intravenously in greater dilution than that supplied by the makers the risks of local thrombosis and of hæmolysis are less. Torrens, Edwards and Wood<sup>33</sup> used a total dosage of 22 g. of myanesin over a period of 9 days (7 g. intravenously and 15 g. intramuscularly) in the treatment of a case of tetanus diluting the solution from the ampoules with an equal amount of sterile distilled water. There was only one instance of local venous thrombosis and this occurred after the one occasion when the myanesin was inadvertently given undiluted. No hæmoglobinuria occurred. The manufacturers state that a 2 per cent. solution of myanesin in normal saline is non hæmolytic but the volume of fluid of this dilution which would have to be injected to produce adequate muscular relaxation in anaesthesia is so great that most anaesthetists consider it impractical.

produced by a different mechanism from that by which *d* tubocurarine chloride acts. The expressions 'curarising or curare like action' should be restricted to those neuromuscular blocking agents which like *d* tubocurarine chloride render the endplate less sensitive to the depolarising action of acetylcholine.

The effect of a fully relaxant dose of C 10 (3 to 5 mg) lasts only 15 to 25 minutes but although full relaxation is of such short duration reasonable operating conditions are present during the phase of respiratory recovery. Further this transient effect is unimportant since repeated doses have no cumulative effects. Dosage with C 10 is remarkably constant 3 to 5 mg being required by most adult patients and there appears to be no sensitivity to it.

Hewer (A J H) Lucas Prescott and Rowbotham<sup>24</sup> who investigated the use of C 10 in anæsthesia state that although previous workers had reported that C 10 caused less respiratory depression than did *d* tubocurarine chloride<sup>25</sup> they found that comparable relaxation could only be obtained with doses which paralysed the muscles of respiration. These investigators consider that C 10 has a wide margin of safety provided adequate pulmonary ventilation is maintained. The action of C 10 is short lasting and the rapidity with which full respiratory exchange is reached once respiratory recovery has begun is very quick.

There is no satisfactory antidote to C 10. Pentamethonium iodide or C 5 (bistrimethylammonium pentane diiodide) will if it is true cause respirations to return but in effective dosage it has unpleasant and serious side effects due to its ganglion blocking action which causes severe lowering of blood pressure. I do not therefore recommend its use as an antidote to C 10.

C 10 has also a use outside anæsthesia. It is considered superior to *d* tubocurarine chloride for modifying electrical convulsions because unlike *d* tubocurarine chloride it is no histamine liberator and therefore does not produce any histamine like reactions and also because muscular paralysis passes off more rapidly<sup>26</sup>. Hewer (A J H) *et al*<sup>24</sup> found that an intradermal injection of 0.1 cc of an 0.1 per cent solution of C 10 did not produce the characteristic histamine wheal and flare which appeared in all of 100 cases similarly tested with a solution of *d* tubocurarine chloride of the same or greater dilution.

### Myanesin

Myanesin is  $\alpha$   $\beta$  dihydroxy  $\gamma$  (2 methyl phenoxy) propane a synthetic relaxant made by British Drug Houses Ltd and is considered last because it differs essentially in its site of action from the relaxants

- (24) Hewer A J H, Lucas B G B Prescott F and Rowbotham E S  
1949 *Lancet* 1, 817
- (25) Organe G Paton W D M and Zaimis E J 1949 *Lancet* 1, 21
- (26) Hobson J A and Prescott F 1949 *Lancet* 1, 819
- (27) Berger F M and Bradley W 1946 *Brit J Pharmacol* 1, 4 265
- (28) Stephen C R and Chandy J 1947 *Canad med Ass J* 57, 463
- (29) Hunter, A R and Waterfall J M 1948 *Lancet* 1, 366
- (30) Davison M H A 1948 *Brit med J* 1, 544
- (31) Mallinson F B 1947 *Lancet* 1, 98
- (32) Clendon D R T and Penfold J B 1949 *Lancet* 2 987
- (33) Torrens J A Edwards P M and Wood M W W 1948 *Lancet*  
2 807

Read also

- Feldberg W 1951 *Brit med J* 1, 967
- Paton W D M 1951 *Brit med J* 1, 773



The possible dangers of intravascular hæmolysis are anuria and permanent renal damage and myanesin should not be used in cases of impaired renal function. The evidence that myanesin causes anuria is still controversial and is not conclusive and there is no evidence that it can cause permanent renal damage but it is wise to use this drug in the smallest effective dosage \*\*

For anæsthesia the dose of myanesin equivalent to a 15 mg dose of *d* tubocurarine chloride is 1 g (10 c c). The antidote to myanesin is not prostigmin but picrotoxin but I have never needed to use an antidote when I have used myanesin as a relaxant in anæsthesia since the respiratory depression which occurs is negligible.

Myanesin is also effective if given by mouth and the makers supply an elixir which is recommended for the treatment of Parkinsonism of various spastic conditions such as spastic hemiplegias and paraplegias of the lower limbs due to cerebral hæmorrhage, disseminated sclerosis and other upper motor neurone lesions and of the spastic and other types of cerebral diplegia.

## REFERENCES

- (1) Waterton C. 1812 *Wanderings in South America* (First published in 1812 see now Everyman's Series) Dent London
- (2) King H. 1935 *J Chem Soc* 1381
- (3) Gill R C. 1940 *White Water and Black Magic* New York
- (4) Griffith H R and Johnson G E. 1942 *Anesthesiology* 3 418
- (5) Wintersteiner O and Dutcher J D. 1943 *Science* 97, 467
- (6) Griffith H R. 1945 *J Amer med Ass* 127, 642
- (7) Griffith H R. 1945 *Lancet* 2 74
- (8) Griffith H R. 1945 *Canad med Ass J* 52, 391
- (9) Gray I C and Halton J. 1946 *Proc Roy Soc Med* 39 400
- (10) Bernard C. 1850 *Compt rend Soc de biol* 2 195
- (11) Burns B D and Paton W D M. 1950 *Proc XI Int physiol Cong* 136
- (12) West R. 1935 *Proc Roy Soc Med* 28 265
- (13) Cole F. 1946 *Anesthesiology* 7, 190
- (14) Prescott F. 1947 *Proc Roy Soc Med* 50 593
- (15) Cullen S C. 1943 *Surgery* 14 261
- (16) Cullen S C. 1944 *Anesthesiology* 5, 166
- (17) Prescott F, Organe G and Rowbotham S. 1946 *Lancet* 2, 80
- (18) Gray T C. 1947 *Brit med J* 1, 444
- (19) Buller A J and Young I M. 1949 *J Physiol* 109 412
- (20) Hobson J A and Prescott F. 1947 *Brit med J* 1 445
- (21) Macintosh R R. 1949 *Brit med J* 1, 852
- (22) Mushin W W, Wren R, Mason D F J and Langston G T. 1949 *Lancet* 1, 726
- (23) Paton W D M and Zaimis E J. 1950 *Lancet* 2 568

The anæsthetist's apparatus also should be ready. It is often inspected by the patient from sheer curiosity and should be immaculate always. Before it is brought into the dental surgery empty cylinders should have been replaced by full ones, and it should never be necessary to change cylinders in the presence of the patient. Cylinder valves should work smoothly and noiselessly and the administrator should be certain that he has a sufficient supply of nitrous oxide and oxygen. The anæsthetist's instruments should have been sterilised after his previous case and although they need not necessarily remain sterile they should be clean and ready for immediate use. When he enters the dental surgery the anæsthetist's only necessary preparations should be to open his bag and to wash his hands.

Impressions made by one person on another in ordinary civil life are apt to be accentuated at such a time as this. After the introductions have been made, the anæsthetist should use any device he thinks legitimate to remove a patient's apprehension although if he is inexperienced in dental anæsthesia he may be far from feeling the confidence which it is necessary for him to instil into the patient. He should make every effort to show the patient that he has control of the situation. Although the anæsthetist should be gentle and kind it is a wise policy to be firm. Anticipated difficulties should not be discussed: the magnitude of the surgical undertaking can be dilated upon after its achievement, not before the anæsthetic is commenced!

With the object of ensuring that all impressions made on a patient shall be pleasing some anæsthetists apply scent to the nose-piece. Certain patients particularly children or nervous women find that the smell of the scent is welcome and helps to divert attention to happier thoughts. On the other hand a few strongly object to scent: some indeed dislike it even more than any other feature of the anæsthetic. Once scent has been applied to a nose- or face-piece it is almost impossible to dispel all traces of it and for this reason we have abandoned its use for the sake of patients who find scent repulsive.

The anæsthetist must be prepared to find some feature about which his patient is sensitive and must take special care not to draw attention to it. For instance, a surprising number of people wear wigs and as a rule the wearer of a wig is extremely conscious of it and fears that it will be disturbed or obviously noticed. The dental anæsthetist therefore should take particular care when applying the nose-piece that neither he nor any part of the apparatus should touch the wig. A

## CHAPTER XXIII

## PRELIMINARY APPROACH TO DENTAL ANÆSTHESIA

THE preparations preliminary to dental anæsthesia considered here should be observed in every case whatever anæsthetic is to be given. The importance of elimination of apprehension in the attainment of smooth anæsthesia with nitrous oxide has already been dealt with. By the time the patient arrives at the dental surgery premedication if any has already been given. For an ambulatory patient this will be only mild and attention to details calculated to put the patient at his ease constitutes psychological treatment which is of great additional value.

The punctuality of all concerned in the operation is important. The patient should be told to arrive in plenty of time. If he leaves his business late and rushes to have his tooth out he is always flustered. If delayed in a traffic block he develops exaggerated irritability. It is preferable for him to plan to arrive ten minutes before the extraction is due so that he will have an opportunity to sit down, to relax and to read his out-of-date *Punch*. The dentist and anæsthetist also should make a point of punctuality. A nervous subject may work himself into a panic if he is kept waiting after the time appointed for the operation and delay may easily give the patient the impression that his operation is not considered important.

Before the patient is shown into the dental surgery he should be given an opportunity to empty his bladder. During times of mental tension, urine is secreted freely, and the discomfort of a full bladder may add to a patient's anxiety. This precaution is also of practical importance since during anæsthesia the bladder if full may be emptied involuntarily.

The dental work to be done should have been determined if possible at a sitting before the day of operation so that time is not spent in making an examination and planning the extent of the extractions on the spur of the moment.

Everything should be ready for the operation before the patient enters the dental surgery. The forceps necessary for the extraction should have been sterilised already and be set ready covered with a sterile towel out of the patient's sight.

The anæsthetist's apparatus also should be ready. It is often inspected by the patient from sheer curiosity and should be immaculate always. Before it is brought into the dental surgery empty cylinders should have been replaced by full ones, and it should never be necessary to change cylinders in the presence of the patient. Cylinder valves should work smoothly and noiselessly, and the administrator should be certain that he has a sufficient supply of nitrous oxide and oxygen. The anæsthetist's instruments should have been sterilised after his previous case and although they need not necessarily remain sterile they should be clean and ready for immediate use. When he enters the dental surgery the anæsthetist's only necessary preparations should be to open his bag and to wash his hands.

Impressions made by one person on another in ordinary civil life are apt to be accentuated at such a time as this. After the introductions have been made the anæsthetist should use any device he thinks legitimate to remove a patient's apprehension although if he is inexperienced in dental anæsthesia he may be far from feeling the confidence which it is necessary for him to instil into the patient. He should make every effort to show the patient that he has control of the situation. Although the anæsthetist should be gentle and kind it is a wise policy to be firm. Anticipated difficulties should not be discussed: the magnitude of the surgical undertaking can be dilated upon after its achievement, not before the anæsthetic is commenced!

With the object of ensuring that all impressions made on a patient shall be pleasing, some anæsthetists apply scent to the nose-piece. Certain patients particularly children or nervous women, find that the smell of the scent is welcome and helps to divert attention to happier thoughts. On the other hand a few strongly object to scent, some indeed dislike it even more than any other feature of the anæsthetic. Once scent has been applied to a nose- or face-piece, it is almost impossible to dispel all traces of it, and for this reason we have abandoned its use for the sake of patients who find scent repulsive.

The anæsthetist must be prepared to find some feature about which his patient is sensitive and must take special care not to draw attention to it. For instance a surprising number of people wear wigs and as a rule the wearer of a wig is extremely conscious of it and fears that it will be disturbed, or obviously noticed. The dental anæsthetist therefore should take particular care when applying the nose-piece that neither he nor any part of the apparatus should touch the wig. A

patient's awareness of his wig is often revealed during recovery from anæsthesia, when almost invariably his first action often before full consciousness is attained is to raise his hand to adjust it

As soon as the patient is settled in the dental chair he is told to blow his nose so that it will be clear of any obstruction Dentures should then be taken out and any tight clothing loosened A man is usually more at his ease without his collar on these occasions and its removal may at least prevent the embarrassment of his leaving the surgery with a blood spattered collar

### Examination of the Patient

For reasons given (p 363) we do not approve of a routine examination before anæsthesia in the dental surgery Various tests have been devised to assess the functional condition of the heart but the only ones which we find reliable are those which give an indication of the vital capacity of the lungs Cardiac decompensation is accompanied by diminution of the vital capacity which when reduced to 40-70 per cent of the normal is shown clinically by dyspnoea on moderate exercise The vital capacity can be measured by a spirometer but a satisfactory evaluation of it and therefore of cardiac efficiency can be formed more simply by carrying out Henderson's breath holding test.<sup>1 2</sup> After a moderately deep inspiration a healthy subject is able to hold his breath for 45 seconds Inability to hold the breath for longer than 15 seconds should be taken as evidence of cardiac insufficiency of a degree which makes it undesirable that the O percentage in the inhaled mixture should be reduced below 20

### POSITION OF THE PATIENT IN THE DENTAL CHAIR

The patient should be allowed to sit in the attitude he finds most comfortable Provided this is reasonably good it need not be altered since he is more likely to go off to sleep peacefully in a comfortable position than in any other Generally however, it is necessary in order to have his hips well back to ask him to make himself tall in the chair The neck rest should then be adjusted to fit the nape of the neck and the patient's head neck and trunk should be approximately in a straight line The head rest should be firmly fixed since if it slips down the head becomes over extended Any such sudden change in position during the course of operation may lead to operative difficulties and to dangers

When the patient is in this position respiration is free and the head easy to control Notice

1 Hips well back in the chair

2 Head neck and chest in a straight line

3 Head rest fitting the nape of the neck

4 The legs and feet in the position usually adopted when sitting

5 The patient is relaxed

6 The fingers are interlocked As an alternative one hand can be held inside the other

7 A strap is around the pelvis

The position of the legs is not of great importance, provided the patient does not obtain a purchase with them against the foot rest If he sits down with his legs crossed and finds this attitude most comfortable,

there is no real reason for asking him to uncross them Occasionally after this position when consciousness returns the patient finds his leg has gone to sleep this effect is only transient and full recovery of power is attained within a minute or two

A patient sometimes seems at a loss as to what to do with his hands and can be advised to hold one hand inside the other Some anaesthetists think it best with a patient likely to be obstructive to ask him to intertwine his fingers This is suggested with the idea that if after anaesthesia is begun he is disposed to struggle he may have difficulty in disentangling his own fingers A very nervous patient often wishes to hold someone's hand This may give him comfort and should be allowed but the hand should not be the anaesthetist's because when losing consciousness the patient may grip so tightly that instant disengagement of the hand may be impossible The nurse or dentist whose hand is held should know that the patient's



FIG 61

patient's awareness of his wig is often revealed during recovery from anæsthesia when almost invariably his first action often before full consciousness is attained is to raise his hand to adjust it

As soon as the patient is settled in the dental chair he is told to blow his nose so that it will be clear of any obstruction. Dentures should then be taken out and any tight clothing loosened. A man is usually more at his ease without his collar on these occasions and its removal may at least prevent the embarrassment of his leaving the surgery with a blood spattered collar

### Examination of the Patient

For reasons given (p 363) we do not approve of a routine examination before anæsthesia in the dental surgery. Various tests have been devised to assess the functional condition of the heart but the only ones which we find reliable are those which give an indication of the vital capacity of the lungs. Cardiac decompensation is accompanied by diminution of the vital capacity which when reduced to 40-70 per cent of the normal is shown clinically by dyspnoea on moderate exercise. The vital capacity can be measured by a spirometer but a satisfactory evaluation of it and therefore of cardiac efficiency can be formed more simply by carrying out Henderson's breath holding test<sup>1 2</sup>. After a moderately deep inspiration a healthy subject is able to hold his breath for 45 seconds. Inability to hold the breath for longer than 15 seconds should be taken as evidence of cardiac insufficiency of a degree which makes it undesirable that the O<sub>2</sub> percentage in the inhaled mixture should be reduced below 20

### POSITION OF THE PATIENT IN THE DENTAL CHAIR

The patient should be allowed to sit in the attitude he finds most comfortable. Provided this is reasonably good it need not be altered since he is more likely to go off to sleep peacefully in a comfortable position than in any other. Generally however it is necessary in order to have his hips well back to ask him to make himself tall in the chair. The neck rest should then be adjusted to fit the nape of the neck and the patient's head, neck and trunk should be approximately in a straight line. The head rest should be firmly fixed since if it slips down the head becomes over extended. Any such sudden change in position during the course of operation may lead to operative difficulties and to dangers

unnecessary but should be used always when it might prove of value. Often the difficulties which will be encountered either in anæsthesia or in the operation are under-estimated and anæsthesia has unexpectedly to be prolonged. In such cases if a strap has been applied as a routine measure the help it gives will be found invaluable.

The patient's permission to apply a strap should not be asked but the reason for its use should be explained to him. As soon as he is in a good position the anæsthetist tells him that he is to be given a peaceful anæsthetic during which he will relax and therefore will tend to slip. To prevent this he will be supported by a strap round the hips.



FIG 63 — Strap loosely applied before induction. The strap is placed round the pelvis so that when it is tightened it does not restrict respiration.



FIG 64 — Strap tightened after anæsthesia is established.

The strap should not be tight while the patient is conscious but as soon as consciousness is lost and before the dentist begins to operate it should be tightened firmly.

If the application of the strap is managed properly the patient rarely protests against it. If there is any objection it is much better to let the patient have his own way and to dispense with it, since otherwise he may feel that he is being forcibly restrained. This idea may rouse antagonism and make him liable to struggle during induction. The application of a strap here can be deferred until the patient is unconscious but it must be remembered that some patients become



grip may become tight enough to be very painful, and that a female patient with sharp fingernails may dig them into the hand she holds

### Use of a Strap

The use of a strap is not a confession of weakness but a sign of experience. There are two conditions in which it should be regarded as essential

(1) For the difficult patient the so called anæsthetic resistant type who is apt to struggle during light anæsthesia and may then slip into a position inconvenient for the operator (fig 62) or develop opisthotonos (fig 26 p 116) if a strap is not used and (2) for a dental operation which it is expected will be difficult or prolonged to whatever type the patient may belong



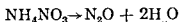
FIG 62

If a strap is not used the patient may slip down in the chair. Valuable time and energy will be spent in hoisting him into position again and in holding the jaw forward and up to prevent respiratory obstruction (p 295) and to allow the dentist access to the mouth. The anæsthetist's hands soon become exceedingly tired and neither hand is free to mop the site of operation nor to make any adjustments to apparatus. When the patient's pelvis is fixed by the strap he is so steadied that his head can easily be kept in position on the head rest and although he may struggle or relax he will not slip down in the chair. In the great majority of cases the strap is entirely

## CHAPTER XXIV NITROUS OXIDE

### PHYSICAL PROPERTIES

NITROUS oxide ( $N_2O$  laughing gas ) is a colourless gas with a faint not unpleasant smell and a slightly sweet taste The gas has a density of 1.5, i.e. it is one and a half times as heavy as air It is prepared by heating ammonium nitrate to  $250^\circ C$



After removal of impurities the gas is dried with great care compressed and filled into cylinders Nitrous oxide condenses to form a colourless liquid at  $0^\circ C$  at a pressure of 30 atmospheres The pressure in the cylinders at normal room temperature is 650 lb. per square inch (= 40 atmospheres) A pressure gauge attached to a cylinder of  $N_2O$  to show the pressure of the gas within is no guide to the amount which remains in the cylinder, because the pressure will remain constant until all the liquid is vaporised when if the valve of the cylinder is left open the pressure suddenly falls to zero An accurate estimation of the quantity of  $N_2O$  remaining in a cylinder can be made only by weighing the cylinder and contents Fifty gallons of  $N_2O$  weigh 15 ozs Since its weight when empty and full is stamped on every cylinder it is simple to calculate the  $N_2O$  content The stamp on the cylinder illustrated shows that when empty (E) it weighs 15 lb 13 ozs and when full (F) 19 lb 9 ozs If a balance is not available a very rough estimate

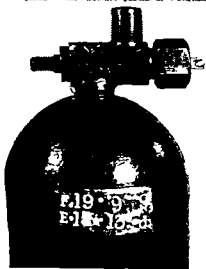


FIG. 65

turbulent early and slip down before there is an opportunity to put on the strap

As soon as the patient is properly settled the teeth to be extracted are pointed out and then the mouth inspected for crowns or other delicate structures so that these will not be injured if a Mason's gag is used. The prop is then put into position care being taken not to open the mouth too widely since this makes the patient uncomfortable and nasal respiration difficult (p. 296). Adjustment of the chair should be made either before the anæsthetic is begun or after the patient is anæsthetised. Movement of the chair during induction is liable to initiate mental disturbance which may result in violence.

Beyond telling the patient to blow the gas away through your nose it is a mistake to give him specific instructions as to how he should breathe. If he asks for instructions however these should be given and they should be simple and definite. He is told to breathe perfectly naturally and that the anæsthetist will adjust the anæsthetic to the respirations. No impression of hurry should be given at any time during the procedure but time should not be lost unnecessarily before beginning anæsthesia.

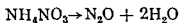
#### REFERENCES

- (1) Clement F. W. 1939 *Nitrous Oxide Oxygen Anesthesia* London 45
- (2) Macleod J. J. R. 1935 *Physiology in Modern Medicine* London 331

## CHAPTER XXIV NITROUS OXIDE

### PHYSICAL PROPERTIES

NITROUS oxide ( $\text{N}_2\text{O}$  laughing gas) is a colourless gas with a faint, not unpleasant smell and a slightly sweet taste. The gas has a density of 1.5, i.e. it is one and a half times as heavy as air. It is prepared by heating ammonium nitrate to  $250^\circ\text{C}$ .



After removal of impurities the gas is dried with great care compressed and filled into cylinders. Nitrous oxide condenses to form a colourless liquid at  $0^\circ\text{C}$  at a pressure of 30 atmospheres. The pressure in the cylinders at normal room temperature is 650 lb. per square inch (= 40 atmospheres). A pressure gauge attached to a cylinder of  $\text{N}_2\text{O}$  to show the pressure of the gas within is no guide to the amount which remains in the cylinder, because the pressure will remain constant until all the liquid is vaporised when if the valve of the cylinder is left open the pressure suddenly falls to zero. An accurate estimation of the quantity of  $\text{N}_2\text{O}$  remaining in a cylinder can be made only by weighing the cylinder and contents. Fifty gallons of  $\text{N}_2\text{O}$  weigh 15 ozs. Since its weight when empty and full is stamped on every cylinder it is simple to calculate the  $\text{N}_2\text{O}$  content. The stamp on the cylinder illustrated shows that when empty (E) it weighs 15 lb 13 ozs. and when full (F) 19 lb 9 ozs. If a balance is not available a very rough estimate

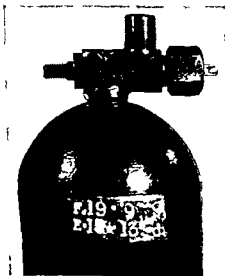


FIG. 65

turbulent early and slip down before there is an opportunity to put on the strap

As soon as the patient is properly settled the teeth to be extracted are pointed out and then the mouth inspected for crowns or other delicate structures so that these will not be injured if a Mason's gag is used. The prop is then put into position care being taken not to open the mouth too widely since this makes the patient uncomfortable and nasal respiration difficult (p. 296). Adjustment of the chair should be made either before the anæsthetic is begun or after the patient is anæsthetised. Movement of the chair during induction is liable to initiate mental disturbance which may result in violence.

Beyond telling the patient to blow the gas away through your nose it is a mistake to give him specific instructions as to how he should breathe. If he asks for instructions however these should be given and they should be simple and definite. He is told to breathe perfectly naturally and that the anæsthetist will adjust the anæsthetic to the respirations. No impression of hurry should be given at any time during the procedure but time should not be lost unnecessarily before beginning anæsthesia.

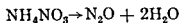
#### REFERENCES

- (1) Clement F. W. 1939 *Nitrous Oxide Oxygen Anesthesia* London 45
- (2) Macleod J. J. R. 1935 *Physiology in Modern Medicine* London 331

## CHAPTER XXIV NITROUS OXIDE

### PHYSICAL PROPERTIES

NITROUS oxide ( $\text{N}_2\text{O}$  laughing gas ) is a colourless gas with a faint, not unpleasant smell and a slightly sweet taste The gas has a density of 1.5, i.e. it is one and a half times as heavy as air It is prepared by heating ammonium nitrate to  $250^\circ\text{C}$



After removal of impurities the gas is dried with great care compressed and filled into cylinders Nitrous oxide condenses to form a colourless liquid at  $0^\circ\text{C}$  at a pressure of 30 atmospheres The pressure in the cylinders at normal room temperature is 650 lb. per square inch (= 40 atmospheres) A pressure gauge attached to a cylinder of  $\text{N}_2\text{O}$  to show the pressure of the gas within is no guide to the amount which remains in the cylinder, because the pressure will remain constant until all the liquid is vaporised when if the valve of the cylinder is left open the pressure suddenly falls to zero An accurate estimation of the quantity of  $\text{N}_2\text{O}$  remaining in a cylinder can be made only by weighing the cylinder and contents Fifty gallons of  $\text{N}_2\text{O}$  weigh 15 ozs Since its weight when empty and full is stamped on every cylinder it is simple to calculate the  $\text{N}_2\text{O}$  content The stamp on the cylinder illustrated shows that when empty (E) it weighs 15 lb 13 ozs and when full (F) 19 lb 9 ozs If a balance is not available a very rough estimate

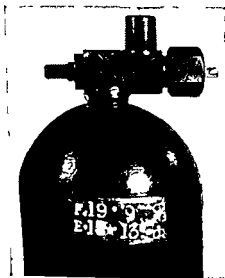


FIG 65

of the cylinder contents may be obtained by tapping the cylinder with a metal spanner. An empty cylinder gives out a high pitched note one containing liquid  $N_2O$  a dull note

In contrast, it should be noted that oxygen in cylinders is in a gaseous state. In this country oxygen cylinders are filled to a pressure of about 120 atmospheres, or 1,800 lb. per square inch. A pressure gauge indicates accurately the amount still remaining in an oxygen cylinder since, as the oxygen flows away, the gaseous pressure falls. The oxygen content of a cylinder therefore can be estimated if its capacity when full and the pressure of oxygen within it are known. Thus if the pressure of a cylinder which contains when full 30 gallons of oxygen registers 40 atmospheres pressure the contents of the cylinder will be  $\frac{40}{120} \times 30 = 10$ , gallons

## GENERAL CONSIDERATIONS

Nitrous oxide the first anæsthetic to be discovered has retained its popularity because of its safety the quick recovery and lack of after effects which follow its use and because it is the only inhalation anæsthetic in common use which is non explosive. The safeness of  $N_2O$  is dependent upon its lack of potency. It is the only anæsthetic which is administered with a lower percentage of oxygen than exists in the atmosphere. Nitrous oxide given with 20 per cent of oxygen will not only not cause death but will scarcely produce unconsciousness. In contrast thiopentone can cause death even when a patient is breathing 100 per cent oxygen so can ether vapour or cyclopropane administered with over 50 per cent oxygen. To anæsthetise an average patient with  $N_2O$  alone a certain amount of asphyxia must be deliberately induced and indeed overdosage is nothing more nor less than an undesirable degree of asphyxia. The sight of a cyanosed and possibly jactitating patient is so alarming that it prevents even the inexperienced anæsthetist from being too venturesome with this anæsthetic. Quick recovery after  $N_2O$  anæsthesia and freedom from after effects are due to the rapidity with which the oxygen lack element of the anæsthetic is alleviated as soon as the mask is removed from the face.

Useful as  $N_2O$  is it has its limitations and these are imposed also by the factor responsible for its safeness i.e. its lack of potency. This gas alone cannot provide good muscular relaxation nor is it potent enough to subdue a markedly anæsthetic resistant patient unless he has been strongly premedicated or unless the oxygen intake is reduced so much that asphyxial movements make operating difficult. Never

theless  $N_2O$  is quite suitable for almost all minor operations and is the anæsthetic of choice for dental surgery in the ambulatory patient

Successful administration of  $N_2O$  depends largely on the experience and technical skill of the anæsthetist but proper premedication is also important and sometimes supplementary anæsthesia with thiopentone, hexobarbitone or ethyl chloride is necessary. Success certainly does not depend upon the use of any particular machine. Perusal of commercial advertising literature suggests that the most important factor is to purchase a certain machine after which all that remains to be done is to apply the nose piece adjust the oxygen percentage as directed, turn on the flow of gases and allow the dentist to proceed. Some makers state that a required depth of anæsthesia can be obtained by giving a specified mixture of  $N_2O$  and  $O_2$  for a prescribed length of time. It is disappointing that the apparatus does not include an automatic forceps which would deal equally well with loose incisors and impacted wisdom teeth! Reliance should not be placed on a machine merely because it is accurate elaborate and expensive. Even the best machines are only a mechanical convenience to the anæsthetist. In the dental surgery good anæsthesia can be obtained by the use of an apparatus consisting of a cylinder of gas a rebreathing bag rubber tubing and a nose piece. If the anæsthetist cannot obtain satisfactory results using such apparatus it is very unlikely that he will obtain better by means of the more complicated machines on the market.

This book refers to the use of  $N_2O$  only in minor surgery, and the particular emphasis laid upon the weakness of the anæsthetic properties of this agent may be thought to imply that it can never be used successfully for major surgery. Such use is indeed possible but to obtain good results the effect of  $N_2O$  must be reinforced by powerful premedication by basal anæsthesia, or by some other method of reducing the patient to a C3 condition e.g. by the secondary saturation technique of McKesson<sup>1</sup>. The use of  $N_2O$  for major surgery is fraught with disappointment if the technique is not carried out boldly and if the anæsthetist is inexperienced with danger if it is.

The essentials of the use of  $N_2O$  for minor surgery will be dealt with here by describing its application in the dental surgery.

### APPARATUS FOR USE IN THE DENTAL SURGERY

The apparatus described in this chapter is that which we have used for some years both in private and hospital practice.

The handle of Macintosh's bag is placed out of centre so that the



of the cylinder contents may be obtained by tapping the cylinder with a metal spanner. An empty cylinder gives out a high pitched note one containing liquid  $N_2O$  a dull note

In contrast it should be noted that oxygen in cylinders is in a gaseous state. In this country oxygen cylinders are filled to a pressure of about 120 atmospheres, or 1,800 lb. per square inch. A pressure gauge indicates accurately the amount still remaining in an oxygen cylinder since, as the oxygen flows away, the gaseous pressure falls. The oxygen content of a cylinder therefore can be estimated if its capacity when full and the pressure of oxygen within it are known. Thus if the pressure of a cylinder which contains when full 30 gallons of oxygen registers 40 atmospheres pressure the contents of the cylinder will be  $\frac{40}{120} \times 30 = 10$  gallons

## GENERAL CONSIDERATIONS

Nitrous oxide the first anæsthetic to be discovered has retained its popularity because of its safety the quick recovery and lack of after effects which follow its use and because it is the only inhalation anæsthetic in common use which is non explosive. The safeness of  $N_2O$  is dependent upon its lack of potency. It is the only anæsthetic which is administered with a lower percentage of oxygen than exists in the atmosphere. Nitrous oxide given with 20 per cent of oxygen will not only not cause death but will scarcely produce unconsciousness. In contrast thiopentone can cause death even when a patient is breathing 100 per cent oxygen so can ether vapour or cyclopropane administered with over 50 per cent oxygen. To anæsthetise an average patient with  $N_2O$  alone a certain amount of asphyxia must be deliberately induced and indeed overdosage is nothing more nor less than an undesirable degree of asphyxia. The sight of a cyanosed and possibly jactitating patient is so alarming that it prevents even the inexperienced anæsthetist from being too venturesome with this anæsthetic. Quick recovery after  $N_2O$  anæsthesia and freedom from after effects are due to the rapidity with which the oxygen lack element of the anæsthetic is alleviated as soon as the mask is removed from the face.

Useful as  $N_2O$  is it has its limitations and these are imposed also by the factor responsible for its safeness i.e. its lack of potency. This gas alone cannot provide good muscular relaxation nor is it potent enough to subdue a markedly anæsthetic resistant patient unless he has been strongly premedicated or unless the oxygen intake is reduced so much that asphyxial movements make operating difficult. Never-

is lifted the apparatus is withdrawn from behind the leather partition

After the tray is replaced the apparatus is ready for use These preparations can be completed within ten seconds of the side catches being released

The contents of the tray are

- 1 set of 6 Trewby props
- 2 de Pass props
- 1 central swivel prop
- 2 Fergusson gags
- 6 average size bleached coarse-meshed, marine sponges with tapes attached
- 6 mouth-packs
- 1 tongue-forceps
- 2 bottles of ethyl chloride

The oxygen bag is contained within the bag for  $N_2O$  Unless the  $O_2$  bag is over distended the pressures within the two bags will be approximately the same

When the handle of the Macintosh's mixing chamber<sup>3</sup>

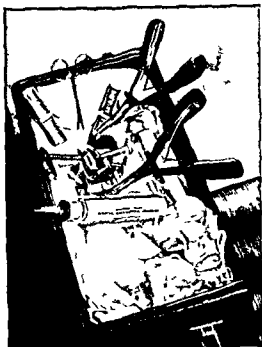


FIG 70

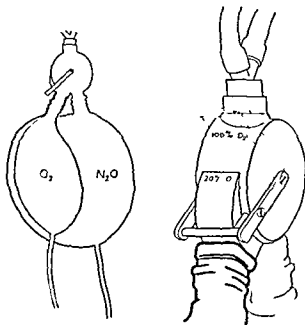


FIG 71—Macintosh's mixing chamber

is in the position illustrated the  $O_2$  bag is cut off and the  $N_2O$  port is fully open As the handle is moved upwards, an increasing percentage of  $O_2$  is admitted When the handle is in the half-way position 20 per cent oxygen is admitted and in its uppermost position (dotted line) the  $O_2$  port is fully open and that of  $N_2O$  closed This arrangement permits fine graduation of the  $O_2$  scale up to 20 per cent



FIG 66



FIG 67

bag will balance when lifted. When the two side catches are released and the lid raised, the front face to which the cylinders are attached falls down.



FIG 68



FIG 69

The case is equipped with three  $N_2O$  cylinders each holding 50 gallons and one  $O_2$  cylinder of 15 gallons capacity. When the tray

hot pipes or furnaces because any rise of temperature may increase the internal pressure so much that they may burst. Although the valve will not leak if it has been properly turned off a  $N_2O$  cylinder which has been stored for some time should be weighed before use to verify that it has not become empty as a result of a slow leakage.

**Advice on the use of the apparatus**—Before anæsthesia is begun the anæsthetist should make it part of his routine practice to observe the following points

1 The apparatus should be inspected to be sure that it is spotlessly clean that the bag and cylinders are properly connected, and that the equipment in the tray is complete

2 Anæsthesia should never be begun when a cylinder in the apparatus is known to be empty. The anæsthetist should be satisfied he has sufficient gas to conclude the anæsthetic. Fear that the gas may run short disturbs the anæsthetist's self-assurance and an attempt to economise gas may ruin an anæsthetic.

3 At the end of an operation any cylinder which has been emptied should immediately be replaced by a full one. The apparatus is then always ready for use on another case.

4 Full cylinders when delivered have a complete paper band gummed round the middle. When an empty cylinder is replaced by a full one the band is at once scraped off the one in use.

5 The anæsthetic should always be begun from the cylinder partly used—i.e. from the one with the torn band—and this cylinder used until it is empty. In the apparatus illustrated (fig 69) the anæsthetist then always has over 100 gallons of gas available at the beginning of every operation.

6 The fibre washers between the cylinder and the gas union become compressed and lose their resiliency through constant use. They should be changed from time to time to prevent leakage of gas when the cylinder is turned on.

7 The footkeys of the cylinders should be turned slowly to verify that the valves are working sweetly. A gentle flow of gas should emerge from the nose-piece before this is put over the patient's nose. It is embarrassing to adjust the nose piece then to turn on the footkey and find that gas does not escape until as one continues to turn the key a large flow of gas suddenly commences with much noise.

8 The position of the indicating handle on the mixing chamber should be verified. If it has been left after the previous case in a position to deliver 100 per cent oxygen and its position is not changed before the  $N_2O$  supply is turned on the bag may become very distended with  $N_2O$  which cannot escape before the error is noticed.

Trewby's nose-piece is made of metal. A rubber rim may be attached but we do not use this since some patients find the smell of it objectionable. Care must be taken not to press the nose piece



FIG 72



FIG 73

down heavily on the bridge of the nose because this may cause pain after recovery. The mouth piece should be of rubber so that a gas tight junction can be ensured even in patients with hollow cheeks. The nose piece is in permanent communication with the mixing chamber but the inlet to the mouth piece is automatically closed or opened as the latter is rotated away from the mouth or forwards to cover it. The mouth should be covered only if the patient is mouth breathing. Once nasal respiration is established (p. 225) the mouth piece is rotated away leaving the mouth free for the operator.

**The cylinders**—The cylinder valve may have either a coarse or a fine thread. When a cylinder the valve of which has a coarse thread is turned on the gas may rush out with an explosive noise disconcerting to the patient operator and anaesthetist. Valves with fine threads (p. 334) are essential if a smooth flow of gas is to be obtained. With these the foot can easily control the rate of issue of the gas from the cylinder. Nitrous oxide and oxygen do not deteriorate with keeping but cylinders of these gases should be stored in a place remote from

remembered that the patient's response to an anæsthetic mixture is not instantaneous. The latent period must be allowed for. In nitrous-oxide anæsthesia it is about 10 seconds. Even if oxygen or air is given instead of  $\text{N}_2\text{O}$  at a time when the patient is already cyanosed, he will continue to show signs of oxygen lack for about 10 seconds, i.e. until the oxygen has diffused into the capillaries of the lungs and has circulated peripherally. There is a similar lag in the other direction. Hence if oxygen is continued until signs of light anæsthesia appear even though the supply of  $\text{O}_2$  is then cut off anæsthesia will lighten further sometimes embarrassingly because of the  $\text{O}_2$  still in the lungs. The anæsthetist therefore has to learn to forecast at any time what the patient's condition will be in a further 10 seconds and to make suitable adjustments to meet an oncoming change. For instance, during induction of anæsthesia he must not wait to introduce  $\text{O}_2$  until asphyxial signs appear, but must add a suitable quantity at a time he considers to be about 10 seconds before asphyxial signs would develop if the unchanged mixture were continued. The same principle is applied when signs of lightening anæsthesia appear.

This of course does not apply when the anæsthetist gives oxygen because he is alarmed about the patient's condition. Its administration should then be continued until there is evidence of its having entered the circulation.

## PREVENTION OF DISCOMFORT DURING INDUCTION OF ANÆSTHESIA

One of the commonest complaints after an anæsthetic has been given badly is that there was a feeling of suffocation during induction. This exceedingly unpleasant sensation can easily be prevented. There is a common misapprehension that it is due to lack of oxygen in the inspired air. That this is not so is shown by these facts: a degree of oxygen lack which leads in a normal individual to cyanosis can be produced without discomfort by careful administration of  $\text{N}_2\text{O}$  alone and yet a feeling of suffocation can be produced by administering pure  $\text{O}_2$  under pressure. The reader can easily verify for himself the principal causes of a feeling of suffocation. These are as follows:

### 1 Interference with the Mechanics of Respiration

(a) Occlusion of the mouth by the anæsthetist's hand or by a Paterson's mouth-cover, when the patient attempts to mouth-breathe. This practice is indefensible. An apprehensive patient tends naturally

9 The cylinders should be arranged so that they are within easy reach of the left foot. It is difficult to control a footkey from an awkward angle, and an attempt to do so may result in delivery of an uneven flow of gas. In addition the difficulty of turning off or increasing the flow of gas may divert the anæsthetist's attention from the patient to the cylinders.

## COMMON ERRORS IN THE ADMINISTRATION OF NITROUS OXIDE AND THEIR AVOIDANCE

The specialist with considerable experience in anæsthetics encounters various difficulties so often that he learns how they arise and how they may be avoided or overcome. The general practitioner who gives nitrous oxide only occasionally may not recognise the reasons for failure in a particular case and therefore cannot learn much from it. This chapter includes information found to be in our experience of practical value in providing smooth anæsthesia and good working conditions for the dentist.

Failure to maintain an unobstructed airway throughout anæsthesia is the most frequent cause of difficulties with N<sub>2</sub>O as with other agents. Every respiration should be seen or heard. If more than one breath is missed the cause should be investigated and the operation interrupted until normal breathing is resumed. Observance of this rule would prevent the development of most of the alarming situations in N<sub>2</sub>O anæsthesia in the dental surgery.

A beginner often provides swinging anæsthesia i.e. instead of maintaining a steady level of anæsthesia he first so deprives the patient of O<sub>2</sub> that deep cyanosis and jactitation occur and then becoming alarmed by this picture administers too much O<sub>2</sub> thereby making a quick change from deep to very light anæsthesia. Alternation between deep and light anæsthesia continues throughout the operation. Such anæsthesia leads the dentist into difficulties. Jactitating movements hinder his work but if he is in a hurry or is inexperienced he may try to extract because at this stage the patient is obviously unconscious. However when the anæsthetist swings to light anæsthesia the surgical stimuli usually initiate mouth breathing which leads rapidly to still lighter anæsthesia. The beginner then abruptly deprives the patient entirely of O<sub>2</sub> once more until signs of asphyxia reappear.

This error occurs because the anæsthetist does not make changes in the anæsthetic mixture early enough. It can be avoided if it is

replaced by the nose piece and the anæsthetic continued with  $N_2O$  given nasally

(b) Delivery of gas under considerable positive pressure with the nose piece or face piece closely applied and with the expiratory valve closed. This produces a feeling of distension and suffocation independent of the nature of the gas given. Discomfort is especially marked during expiration. The discomfort of expiration against pressure is appreciated when testing the inlet valve of a service respirator. The outlet valve is then occluded and an attempt made to expire. If the inlet valve is functioning satisfactorily, air cannot pass out and expiration is impeded. The suffocating sensation is as marked as from efforts to inspire from a bag already empty.

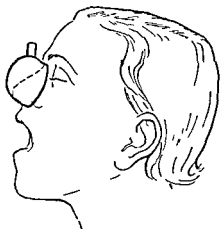


FIG 76



FIG 77

(c) Occlusion of the nares by the lower border of the nose-piece (fig 76). This can occur if the nose-piece is allowed to rise a little instead of being kept down on the upper lip. Good advice to beginners is keep the chin up and the nose-piece down (fig 77).

## 2 Inhalation of Pungent Vapours

A suffocating sensation from this cause is most marked from strong ether vapour given early during the induction of anæsthesia. This factor, which has no application in the administration of  $N_2O$  in the dental surgery need not be further considered here.



to breathe through his mouth during induction of anæsthesia and if he is prevented from doing so and a feeling of suffocation thereby produced, struggling becomes instinctive and a smooth induction impossible. This cause of a feeling of suffocation can be verified by breathing through the mouth and then placing the hand firmly over the open mouth so making attempts at inspiration ineffective. It is well appreciated when testing the efficiency of the outlet valve of a service respirator by occluding the inlet valve and then attempting to inspire. If the valve is efficient the effort is ineffective and provokes an oppressive feeling of suffocation.



FIG 74 —A mouth-cover similar to Paterson's in use. The one way valve allows of expiration and prevents inspiration.



FIG 75 —The mouth covered by the anaesthetist's hand.

*Neither of these methods should ever be used to make a patient breathe through his nose.*

A nose-piece is of course used throughout and for a patient who is mouth breathing a mouth piece should be used which delivers  $N_2O$  so that there is no resistance to his attempts to inspire. Trewby's apparatus (p 216) is the best available. If the administrator has a nose piece but no mouth-piece he can induce with an ordinary full face-piece until surgical anæsthesia is reached and nasal respiration (p 225) established. The face piece then can be

he submits to anæsthesia he feels that he is giving himself into the anæsthetist's care and is grateful for sympathetic reassurance. As induction is begun the anæsthetist both by touch and speech, makes contact with the patient and maintains it until consciousness is lost. During induction the anæsthetist rests his left hand lightly on the patient's left shoulder and says

Now I want you to relax these muscles and then you will go to sleep more comfortably. When this remark is made the shoulder girdle will often drop and the patient relax. This change is commonly seen in the nervous patient who sits down in the dental chair with his muscles taut.

During induction the patient is particularly suggestible. The anæsthetist can encourage the idea of relaxation which should be instilled from the beginning and should repeatedly assure the patient that he is doing well. Anæsthesia usually can be induced smoothly in a calm patient who sits naturally in a relaxed position in the chair. Similarly it is found that if a patient can be made to relax he becomes more peaceful and induction is correspondingly facilitated. The anæsthetist should keep up in a matter of fact voice an almost continuous monologue calculated to be encouraging. The anæsthetist may feel that his talk is monotonous since he repeats it every time he gives an anæsthetic. It is not however monotonous for the patient who is made to realise that he is having undivided attention. Frequent use of such words as relax, sleep and armchair is an aid in accomplishing peaceful induction. It must be remembered that the patient is undergoing an unfamiliar and sometimes an unpleasant experience which at the back of his mind may well be associated with thoughts of the possibility of death. The anæsthetist should continue the monologue until the patient is sound asleep for if the latter is aware of its cessation he may be disturbed thinking that this indicates that the operation is about to commence. There is a



FIG 79

## 3 Psychological Disturbances



FIG 78

Many people feel suffocated if even a detached face piece which permits free inspiration and expiration, is placed *tightly* over the nose and mouth. For these patients an explanation that there will be no resistance to respiration may not suffice and if anæsthesia is to be induced with  $N_2O$  the nose piece should be held off the nose until consciousness is lost. The best method however is to induce anæsthesia with thiopentone so that nothing need be put over the face until consciousness is lost.

### INDUCTION OF ANÆSTHESIA WITH NITROUS OXIDE GIVEN NASALLY

As goes induction so goes the anæsthetic. This saying emphasises the importance of aiming at smooth induction. It is well known that if a person inadvertently finds himself in an atmosphere devoid of  $O_2$  for instance at the bottom of a well or in a coal mine or at the bottom of a ship he suddenly loses consciousness without any preceding unpleasant sensations. If the air in a room could be replaced either suddenly or gradually by  $N_2O$  the occupants would become unconscious without realising that anything untoward was happening. The perfect induction of anæsthesia as far as possible should be equally smooth. Usually the patient closes his eyes as soon as he is settled and if the nose piece is then held about  $\frac{1}{2}$  inch away from his nose and not lowered until consciousness is lost he may not even realise that he is inhaling the anæsthetic. He then recovers consciousness to express surprise that the operation has been completed because he asserts nothing has yet been put over my face.

However unemotional and self confident a patient may be when

within  $\frac{1}{2}$  inch of the patient's nose. Positive pressure of  $N_2O$  is essential since otherwise the concentration of gas over the nose would not be high enough to produce unconsciousness. Ideally the nose-piece should not touch the face until consciousness is lost because many patients dislike having something put over the face. The slight gap between nose-piece and nose is also of importance, since it allows any excess pressure in the bag to be dissipated and thus prevents the feeling of suffocation which otherwise would arise if the expiratory valve were not adequately open.

Except for a very frail or an anæmic patient it is not desirable to give oxygen until anaesthesia is attained. Nitrous oxide alone is no less pleasant to inhale than a mixture of  $N_2O$  and  $O_2$  and the latter means an unnecessary prolongation of the induction period which may be unpleasant for the patient. It is advisable to hasten the robust individual through the period of induction and potential delirium as rapidly as possible and even after reaching the desired stage of anaesthesia it is wise to continue  $O_2$  deprivation for a few seconds, before lightening anaesthesia to the desired level by addition of  $O_2$  to the inhaled mixture.

## ESTABLISHMENT OF NASAL RESPIRATION

Maintenance of surgical anaesthesia by means of nitrous oxide given nasally allows the dental surgeon free access to the mouth. This method sometimes referred to as continuous gas is now almost universally employed for dental work even when only short anaesthesia is required. If this type of anaesthesia is to be successful the patient's respirations must be entirely nasal. Those unfamiliar with this method frequently express surprise or even disbelief that it is possible to induce and maintain anaesthesia by nasal administration of  $N_2O$  when the patient's mouth is propped open. Induction with a nose piece is in fact just as simple for the anaesthetist as with a face-piece and is far more pleasant for the patient. If mouth breathing occurs and the anaesthetist does not know how to abolish it, the administration will be a failure. He must learn therefore the causes of mouth breathing and how to ensure a reversion to nose breathing should it occur.

Mouth breathing a nervous phenomenon is a response to fear or excitement and is part of the fight or flight mechanism. Fundamentally its purpose is to improve the ventilation of the lungs. Removal of apprehension is therefore an essential measure conducive to nasal respiration. Three facts should be appreciated (1) at rest the average

critical moment in induction of anæsthesia particularly with  $N_2O$  when a little encouragement, a kind word and a pat on the shoulder will make all the difference between struggling and violence and quiet induction. This period may extend only for the time taken by one or two breaths.

If the patient begins to struggle during induction he should be opposed only as much as may be necessary. The nurse should not take the initiative and forcibly hold the patient down before there is need for restraint since struggling then appears as a natural response. In any case restraint need be concentrated only on keeping the patient's hands still so that he cannot reach up and pull the apparatus off his face.

The last sense to be lost during induction of anæsthesia is that of hearing. It is also the first to return during recovery so that unguarded remarks should not be made during induction or as the patient returns to consciousness. Before surgical anæsthesia is established a patient passes through a period during which he may respond to auditory stimuli of which he has afterwards no recollection. For instance if the anæsthetist informs the dentist that he can begin the patient previously motionless may raise his hand as if to indicate that he is not yet unconscious though on recovery he does not remember the remark or his response to it. The existence of this state can be demonstrated experimentally.

Gas is given nasally and an onlooker counts the seconds aloud. The anæsthetist asks at irregular intervals. Can you hear me now? and the subject responds by raising his hand. Considerable discrepancy will be found between the last number the subject remembers hearing and that at which he last responded.

During induction the anæsthetist and dentist should not converse nor should there be any rattling of instruments or obvious preparations for the operation. In particular the dentist must not stand ready over the patient forceps in hand anxious to start at the earliest possible moment and for nervous patients who often keep their eyes open during induction the dentist should make a special point of standing with his back to the patient so that the latter does not feel the dentist is hurried or waiting the earliest opportunity to begin extractions.

Just before induction is begun nitrous oxide is turned on gradually and the pressure in the breathing bag increased until a gentle flow of gas emerges from the nose piece which is lowered to

within  $\frac{1}{2}$  inch of the patient's nose. Positive pressure of  $N_2O$  is essential since otherwise the concentration of gas over the nose would not be high enough to produce unconsciousness. Ideally the nose-piece should not touch the face until consciousness is lost because many patients dislike having something put over the face. The slight gap between nose-piece and nose is also of importance since it allows any excess pressure in the bag to be dissipated and thus prevents the feeling of suffocation which otherwise would arise if the expiratory valve were not adequately open.

Except for a very frail or an anæmic patient, it is not desirable to give oxygen until anæsthesia is attained. Nitrous oxide alone is no less pleasant to inhale than a mixture of  $N_2O$  and  $O_2$  and the latter means an unnecessary prolongation of the induction period which may be unpleasant for the patient. It is advisable to hasten the robust individual through the period of induction and potential delirium as rapidly as possible and even after reaching the desired stage of anæsthesia it is wise to continue  $O_2$  deprivation for a few seconds before lightening anæsthesia to the desired level by addition of  $O_2$  to the inhaled mixture.

## ESTABLISHMENT OF NASAL RESPIRATION

Maintenance of surgical anæsthesia by means of nitrous oxide given nasally allows the dental surgeon free access to the mouth. This method sometimes referred to as continuous gas is now almost universally employed for dental work even when only short anæsthesia is required. If this type of anæsthesia is to be successful the patient's respirations must be entirely nasal. Those unfamiliar with this method frequently express surprise or even disbelief that it is possible to induce and maintain anæsthesia by nasal administration of  $N_2O$  when the patient's mouth is propped open. Induction with a nose piece is in fact just as simple for the anæsthetist as with a face-piece and is far more pleasant for the patient. If mouth breathing occurs and the anæsthetist does not know how to abolish it, the administration will be a failure. He must learn therefore the causes of mouth breathing and how to ensure a reversion to nose breathing should it occur.

Mouth breathing a nervous phenomenon is a response to fear or excitement and is part of the fight or flight mechanism, fundamentally its purpose is to improve the ventilation of the lungs. Removal of apprehension is therefore an essential measure conducive to nasal respiration. Three facts should be appreciated (1) at rest the average

critical moment in induction of anæsthesia particularly with  $N_2O$  when a little encouragement a kind word and a pat on the shoulder will make all the difference between struggling and violence and quiet induction This period may extend only for the time taken by one or two breaths

If the patient begins to struggle during induction he should be opposed only as much as may be necessary The nurse should not take the initiative and forcibly hold the patient down before there is need for restraint since struggling then appears as a natural response In any case restraint need be concentrated only on keeping the patient's hands still so that he cannot reach up and pull the apparatus off his face

The last sense to be lost during induction of anæsthesia is that of hearing it is also the first to return during recovery so that unguarded remarks should not be made during induction or as the patient returns to consciousness Before surgical anæsthesia is established a patient passes through a period during which he may respond to auditory stimuli of which he has afterwards no recollection For instance if the anæsthetist informs the dentist that he can begin the patient previously motionless may raise his hand as if to indicate that he is not yet unconscious though on recovery he does not remember the remark or his response to it The existence of this state can be demonstrated experimentally

Gas is given nasally and an onlooker counts the seconds aloud The anæsthetist asks at irregular intervals Can you hear me now? and the subject responds by raising his hand Considerable discrepancy will be found between the last number the subject remembers hearing and that at which he last responded

During induction the anæsthetist and dentist should not converse nor should there be any rattling of instruments or obvious preparations for the operation In particular the dentist must not stand ready over the patient forceps in hand anxious to start at the earliest possible moment and for nervous patients who often keep their eyes open during induction the dentist should make a special point of standing with his back to the patient so that the latter does not feel the dentist is hurried or waiting the earliest opportunity to begin extractions

Just before induction is begun nitrous oxide is turned on gradually and the pressure in the breathing bag increased until a gentle flow of gas emerges from the nose-piece which is lowered to

within  $\frac{1}{2}$  inch of the patient's nose. Positive pressure of  $N_2O$  is essential since otherwise the concentration of gas over the nose would not be high enough to produce unconsciousness. Ideally the nose-piece should not touch the face until consciousness is lost because many patients dislike having something put over the face. The slight gap between nose-piece and nose is also of importance, since it allows any excess pressure in the bag to be dissipated and thus prevents the feeling of suffocation which otherwise would arise if the expiratory valve were not adequately open.

Except for a very frail or an anæmic patient, it is not desirable to give oxygen until anæsthesia is attained. Nitrous oxide alone is no less pleasant to inhale than a mixture of  $N_2O$  and  $O_2$  and the latter means an unnecessary prolongation of the induction period which may be unpleasant for the patient. It is advisable to hasten the robust individual through the period of induction and potential delirium as rapidly as possible and even after reaching the desired stage of anæsthesia it is wise to continue  $O_2$  deprivation for a few seconds before lightening anæsthesia to the desired level by addition of  $O_2$  to the inhaled mixture.

## ESTABLISHMENT OF NASAL RESPIRATION

Maintenance of surgical anæsthesia by means of nitrous oxide given nasally allows the dental surgeon free access to the mouth. This method sometimes referred to as continuous gas is now almost universally employed for dental work even when only short anæsthesia is required. If this type of anæsthesia is to be successful the patient's respirations must be entirely nasal. Those unfamiliar with this method frequently express surprise or even disbelief that it is possible to induce and maintain anæsthesia by nasal administration of  $N_2O$  when the patient's mouth is propped open. Induction with a nose piece is in fact just as simple for the anaesthetist as with a face-piece and is far more pleasant for the patient. If mouth breathing occurs and the anaesthetist does not know how to abolish it the administration will be a failure. He must learn therefore the causes of mouth breathing, and how to ensure a reversion to nose-breathing should it occur.

Mouth breathing a nervous phenomenon is a response to fear or excitement and is part of the fight or flight mechanism. Fundamentally its purpose is to improve the ventilation of the lungs. Removal of apprehension is therefore an essential measure conducive to nasal respiration. Three facts should be appreciated (i) at rest the average



person breathes through his nose (ii) in anæsthesia mouth breathing if it occurs at all is seen only during induction or in anæsthesia too light for the operation being performed (iii) when the stage of surgical anæsthesia is reached respiration is entirely nasal. If properly dealt with nine out of ten patients can be persuaded to nose breathe continuously from the beginning. It is not possible to be sure whether the patient is breathing through his nose or not until the nose piece is lowered on to the face.

A clinical observation made by one of us (R R M) is that expiration through the nose indicates that the patient is also inspiring through the nose. We have found this to be a valuable sign. By using a nose piece fitted with a suitable expiratory valve expirations through it can be heard and the anæsthetist can then tell when the patient is breathing in through the nose. Normally a person breathes in and out through his nose. Under emotional stress he may breathe in and out of his mouth or in through his nose and out through his mouth but he never breathes in through his mouth and out through his nose. *The sound of the expiratory valve on the nose-piece in action is therefore an indication that the patient is also inspiring through his nose.*

If at the beginning of induction a patient is told 'Breathe through your nose' he will as often as not take a deep inspiration through the mouth. If however he is told 'Blow the gas away through your nose' he readily complies with this instruction and the nasal expiration is automatically followed by nasal inspiration so that to and fro nasal respiration is quickly established without the patient realising it.

A curious psychological fact is that a patient often fails to co operate in following instructions if told that his own comfort thereby will be increased whereas he is very likely to carry them out if told that they facilitate the anæsthetist's work. Thus 'Now I want you to help me by blowing the gas away through your nose' It makes my work so very much easier if you will do this for me' is more likely to be successful than 'I want you to blow this gas away through the nose' it will make things more comfortable for you.

If the expiratory valve is not heard when the nose piece is lowered it generally indicates that the patient is breathing in and out of his mouth. Mouth-breathing during induction may occur at the beginning when it is often possible to establish nose breathing by giving a few simple instructions or it may occur in an apprehensive patient just as consciousness is lost and he is then unable to respond to instructions. When it occurs early the patient should be told to breathe out through his nose if he obeys nasal respiration will be

established without the use of the mouth-piece. As soon as the patient is carrying out the instructions properly he should be encouraged by being told 'That is magnificent you have made my work so much easier. Now give six blows through the nose.' The expirations are counted 'One, two, three' etc. during which time anaesthesia becomes established.

If the patient is so unco-operative that he does not follow instructions, or if he first follows them but later reverts to mouth breathing just as consciousness is about to be lost the attempt to induce anaesthesia by nasal administration alone should be abandoned. The mouth piece should be applied at once and not removed until surgical anaesthesia is established *when it will be found that respiration is nasal*. Gas should be led to the mouth by means of the mouth-piece as described. As a rule the patient is breathing deeply, so that anaesthesia is rapidly induced. When gas is delivered through the nose and mouth in this way positive pressure should be increased during inspiration to prevent any leakage of air inwards due to the nose- or mouth piece not fitting well. This can be assured by pressing with the knee on the rebreathing bag during inspiration. Any excess of gas is dissipated through the expiratory valve so that the pressure does not become uncomfortably high.

Another method of producing surgical anaesthesia in face of persistent mouth breathing and one specially useful in highly resistant subjects is to leave the nose piece *in situ* and turn up the napkin on the patient's chest so that part of it covers the mouth, and on it to spray ethyl chloride until anaesthesia is established, the patient by then will have reverted to nose breathing whereupon the anaesthetic is continued with the nose-piece only.

When the mouth piece of the Trewby apparatus is applied the expiratory valve on the nose piece is at once heard to be functioning. This noise is then not a sign of nose breathing since the valve serves both nose piece and mouth piece. As soon as the anaesthetist thinks that nasal respiration is re-established the mouth-piece is withdrawn, if the sound of the expiratory valve still continues his opinion is confirmed. If the patient is found still to be breathing through the mouth however the mouth-piece is at once reapplied and anaesthesia deepened until on withdrawing the mouth piece and rotating it out of the way nasal respiration is found to be completely established. Not until then should the operator be allowed to begin.

Although it is seldom necessary to use the mouth-piece the anaesthetist should lose no time in applying it when its use does become necessary. Mouth breathing can occur at any stage of induction if

anything is done which alarms the patient (e.g. if the position of the chair is altered) or it can occur spontaneously in a nervous patient. Once surgical anæsthesia is established, however, whether respiration remains nasal or becomes oral depends on the relation of the depth of unconsciousness to the severity of the surgical stimulus. Nitrous oxide anæsthesia lightens rapidly if too much  $O_2$  is given, and the anæsthetist must be on the watch for any reversion to mouth breathing. Such a change is appreciated only during the administration of an anæsthetic nasally, and it is then of particular importance. If during the course of a dental operation under nasal  $N_2O$  anæsthesia is inadvertently allowed to lighten and the patient begins to breathe through his mouth, he will rapidly regain consciousness as a result of inspiring air. To prevent this the operation must be suspended at once while the mouth is covered with the mouth piece until nasal respiration is re-established, after which, provided anæsthesia is kept deep enough, the operation can be continued indefinitely.

The imminence of mouth breathing during an operation may be indicated by an expiratory wail, by movements of the head or limbs during extraction, or by other signs of light anæsthesia. If mouth breathing has not actually begun it is sometimes possible at this stage by delivering the  $N_2O$  under increased pressure to deepen anæsthesia again without having to stop the operation and cover the mouth. If, however, the dentist continues to apply strong surgical stimuli it may be impossible to prevent the onset of mouth breathing. If he should then have just one more try at the extraction, anæsthesia may lighten sufficiently for the patient to become almost unmanageable or conscious. At the first suggestion of mouth breathing a pause of a few seconds by the operator will allow the patient to be settled to a steady level of anæsthesia again and will make all the difference between a successful anæsthetic and a fiasco. Team work is essential and the dentist must always defer to the anæsthetist's request to suspend the operation until nasal respiration is re-established.

The anæsthetist should always be prepared to deal with mouth breathing either during induction or light anæsthesia, and he should learn to recognise the types of patient who may give trouble in this respect. These are

- 1 The patient who keeps his eyes open when the nose-piece is applied

- 2 The patient who breathes deeply and rapidly at the beginning of induction, often hunching up his shoulders in the process

- 3 The patient who begins nose breathing regularly but then hesitates and takes one or two rapid breaths

4 The patient, generally a woman, who makes an expiratory wail during induction

When any of the above four signs are noticed the anaesthetist must be ready instantly to cover the mouth

The anaesthetist must be prepared to meet occasionally various rarer difficulties which disturb induction even more than does mouth-breathing. These are usually due to extreme apprehension and are often avoidable by premedication. When they arise, the action taken has to be suited to the individual case, for the conduct of which no set rules can be laid down. In some instances it is advisable to postpone the operation and to arrange for ample premedication to be given before the patient comes to the surgery on a later occasion.

The patient, for instance, may move his head away or become hysterical when the nose-piece is applied. The nose-piece should not then be forced on him or a violent struggle may be precipitated or in a nervous woman or excitable child even screaming. At this early stage it is much better not to attempt to adjust the nose piece against the patient's will but to reason with him, showing him how he can assist in making the operation successful.

The patient at first may be co-operative but may attempt to pull the apparatus off after a few breaths have been taken. He may be unconscious already and beyond the stage where he can understand and obey instructions. The anaesthetist must then decide whether to stop and reason with him or to force the anaesthetic on him, the decision being determined by the degree of unconsciousness which the anaesthetist considers the patient has reached.

Sometimes during induction if respiration has been deep and rapid for a sufficient length of time so much carbon dioxide is washed out of the lungs that a period of apnoea follows which may give rise to anxiety if the anaesthetist does not realise its cause and that normal breathing will be resumed as soon as the  $\text{CO}_2$  concentration in the alveolar air rises again to such a level as to stimulate the respiratory centre. Apnoea therefore occurs in patients who breathe quickly and deeply during induction, thinking that this assists the anaesthetist. They should be told there is no hurry and that breathing should be perfectly natural. Unduly rapid respiration occurs also in the very apprehensive patient, generally a woman, who begins early in induction to breathe at the rate of about sixty per minute. Such manifestation of extreme nervousness usually cannot be controlled by reasoning with the patient. As respiration here is entirely oral the mouth must be covered from the beginning, both nose and mouth piece must be applied firmly and the pressure in the bag increased (to prevent air

anything is done which alarms the patient (e.g. if the position of the chair is altered) or it can occur spontaneously in a nervous patient. Once surgical anaesthesia is established however whether respiration remains nasal or becomes oral depends on the relation of the depth of unconsciousness to the severity of the surgical stimulus. Nitrous oxide anaesthesia lightens rapidly if too much  $O_2$  is given and the anaesthetist must be on the watch for any reversion to mouth breathing. Such a change is appreciated only during the administration of an anaesthetic nasally and it is then of particular importance. If during the course of a dental operation under nasal  $N_2O$  anaesthesia is inadvertently allowed to lighten and the patient begins to breathe through his mouth he will rapidly regain consciousness as a result of inspiring air. To prevent this the operation must be suspended at once while the mouth is covered with the mouth piece until nasal respiration is re-established after which, provided anaesthesia is kept deep enough the operation can be continued indefinitely.

The imminence of mouth breathing during an operation may be indicated by an expiratory wail by movements of the head or limbs during extraction or by other signs of light anaesthesia. If mouth breathing has not actually begun it is sometimes possible at this stage, by delivering the  $N_2O$  under increased pressure, to deepen anaesthesia again without having to stop the operation and cover the mouth. If, however the dentist continues to apply strong surgical stimuli it may be impossible to prevent the onset of mouth breathing. If he should then have just one more try at the extraction anaesthesia may lighten sufficiently for the patient to become almost unmanageable or conscious. At the first suggestion of mouth breathing a pause of a few seconds by the operator will allow the patient to be settled to a steady level of anaesthesia again and will make all the difference between a successful anaesthetic and a fiasco. Team work is essential and the dentist must always defer to the anaesthetist's request to suspend the operation until nasal respiration is re-established.

The anaesthetist should always be prepared to deal with mouth breathing either during induction or light anaesthesia and he should learn to recognise the types of patient who may give trouble in this respect. These are

- 1 The patient who keeps his eyes open when the nose-piece is applied

- 2 The patient who breathes deeply and rapidly at the beginning of induction often hunching up his shoulders in the process

- 3 The patient who begins nose breathing regularly but then hesitates and takes one or two rapid breaths

### Nitrous Oxide and Air

We find it convenient during the course of anæsthesia to admit air by pushing the nose piece forward, at the same time reducing the gas pressure in the bag. The amount of air given by this method cannot be measured accurately, but with experience in combining these two actions the anæsthetist finds it easy to maintain long and even anæsthesia.



FIG 80

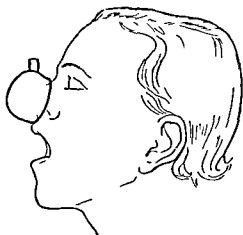


FIG 81

Throughout anæsthesia the nose piece is kept in position by steady-  
ing the tube between thumb and forefinger (fig 80). The jaw is held  
forward with the remaining fingers and the head steadied between  
the palms of the hands. Fig 81 shows the effect of pushing the thumb  
and forefinger forwards thus raising the nose-piece to admit air.  
An advantage of this method is that the amount of  $O_2$  admitted  
can be varied as desired by a small movement of the fingers without  
relaxing the firm hold on the patient's jaw.

### Nitrous Oxide and Oxygen

These two gases can be mixed in any desired proportion by adjusting  
the lever on the mixing chamber (p 215). The nose-piece is kept  
down in position. The disadvantage of this method is that the  
adjustment of the anæsthetic mixture occupies one of the anæsthetist's  
hands at a time when he may want both hands for steadying the head  
or for assisting the dentist in other ways.

The above two methods may be combined with advantage. About

leaking into the circuit) until the patient is deeply unconscious by which time the rate of respiration will have returned to normal. This rapid respiration is as a rule very superficial and does not then lead to apnoea although it will do so if it is at all deep and continues for any length of time.

A small problem but one of practical importance is presented by the patient with a moustache since this may give rise to difficulties because of air leaks during both induction and maintenance of nasal anæsthesia. It is notoriously difficult to get the nose-piece to fit closely on such a patient and the consequent leaking of air alongside it tends to prolong induction and to disturb the maintenance of anæsthesia. Difficulties can be overcome by delivering the gas under positive pressure so that air leaks cannot occur. In fact adequate positive pressure in such a case is the equivalent of an airtight nose piece.

### MAINTENANCE OF ANÆSTHESIA WITH NITROUS OXIDE GIVEN NASALLY

The signs which indicate when the patient is ready to be operated on are described in Chapter VII. Strange as it may seem, it appears necessary to add that crying, screaming and struggling proclaim unmistakably that surgical anæsthesia has not yet been reached!

Oxygen should not be added to the anæsthetic mixture unless the patient is anæmic until surgical anæsthesia is established. Oxygen is then admitted to a sufficient extent to maintain a steady level of anæsthesia. The operation should not be begun immediately surgical anæsthesia is reached since the anæsthetist should first discover by trial roughly what is the correct mixture necessary to maintain anæsthesia at the desired level. To prevent waste of time while this trial is being made the strap can be tightened round the patient's pelvis and the mouth packed off with a sponge or pack.

There is no rule of thumb guide to the mixture of gas and oxygen which a patient should be given. The only guide is his response to the mixture he is already being given. Nitrous oxide with say 12 per cent oxygen may cause respiratory depression in an anæmic person yet may scarcely produce unconsciousness in a plethoric individual. The degree of anoxæmia by which  $N_2O$  must be reinforced to produce unconsciousness no more follows a set rule than does the amount of whisky necessary to produce drunkenness. In both cases the result produced depends on the individual reactions of the patient. Whether nitrous oxide and oxygen or nitrous oxide and air is given is immaterial.

membranes will prevent smooth intubation, some degree of laryngo spasm may develop or the patient may become restless or retch



FIG 82

An accumulation of saliva or blood in the pharynx is best dealt with by leaning the patient forward as far as possible for a few seconds to allow any collection in the mouth to run out. The lower jaw is held forward so that respiration is not interrupted. The posterior pharyngeal wall should not be swabbed, since this may initiate reflex vomiting and in any case will irritate the mucous membrane and lead to renewed secretion of mucus.

If a patient vomits during the course of an operation he should be put into the same position to ensure free drainage from the mouth. It is always a disappointment for him to wake up to find the extractions have not been completed and if the situation is handled correctly the operation need not be abandoned on account of vomiting. The nose piece is removed to allow anaesthesia to lighten sufficiently for the patient to regain his cough reflex and so clear out his mouth. Before consciousness is regained the nose piece is lowered again and very light anaesthesia maintained. As soon as vomiting has finished the patient is placed upright again and the mouth is examined to make sure it is clear of debris a fresh sponge is inserted anaesthesia



6 per cent of  $O_2$  is given continuously with the  $N O$  the nose piece being kept down in position unless more  $O_2$  is required when it can be pushed forward and the pressure of gases reduced so that air is admitted as described above

### Keeping the Airway Clear

This extremely important duty of the anæsthetist is accentuated in every text-book on anæsthesia but the subject is often dismissed without any advice being given to show how it should be accomplished. The necessity for a clear airway in order to ensure an adequate  $O_2$  supply is obvious. Apart from this, inhalation anæsthesia relies on the continuance of normal respiratory movements to carry the anæsthetic drug into the lungs whence, by the process of diffusion it passes into the blood stream. Maintenance of smooth anæsthesia therefore depends on maintenance of a clear airway. In dental anæsthesia the cause *par excellence* of respiratory obstruction is the pushing of the lower jaw backwards or downwards by the dentist so that the tongue is pushed against the posterior pharyngeal wall. This happens most commonly during extraction of lower teeth particularly molars and is only rarely the cause of obstruction during operations on the upper jaw. The common causes of respiratory obstruction in the dental chair their prevention and treatment are discussed in the first few pages of Chapter XXVIII.

It is a counsel of perfection that respiration should not be obstructed even for a single breath but in practice we are in agreement with those who consider that provided the anæsthetist is experienced and can at will correct the obstruction this rule need not always be strictly adhered to. Although we do not recommend the practice we have on occasions deliberately exploited for a few seconds respiratory obstruction caused by the dentist as a means of decreasing the  $O_2$  intake in order to deepen anæsthesia in a robust individual in whom anæsthesia showed signs of lightening. We have also permitted obstruction to persist for one or two breaths in order not to interrupt the dentist when he is having particular difficulty in applying his forceps to a lower molar. In the latter case when the obstruction is relieved, care must be taken that the patient's first breath is one rich in air or oxygen.

Interference with free respiration can occur from mucus or blood gravitating backwards into the pharynx. This is particularly liable to happen if the head is too far extended. If anæsthesia is deep, the mucus or blood may be inhaled during light anæsthesia the irritation they produce on the laryngeal and pharyngeal mucous

membranes will prevent smooth anaesthesia some degree of laryngospasm may develop or the patient may become restless or retch



FIG 82

An accumulation of saliva or blood in the pharynx is best dealt with by leaning the patient forward as far as possible for a few seconds to allow any collection in the mouth to run out. The lower jaw is held forward so that respiration is not interrupted. The posterior pharyngeal wall should not be swabbed, since this may initiate reflex vomiting and in any case will irritate the mucous membrane and lead to renewed secretion of mucus.

If a patient vomits during the course of an operation he should be put into the same position to ensure free drainage from the mouth. It is always a disappointment for him to wake up to find the extractions have not been completed and if the situation is handled correctly the operation need not be abandoned on account of vomiting. The nose piece is removed to allow anaesthesia to lighten sufficiently for the patient to regain his cough reflex and so clear out his mouth. Before consciousness is regained the nose piece is lowered again and very light anaesthesia maintained. As soon as vomiting has finished the patient is placed upright again and the mouth is examined to make sure it is clear of debris a fresh sponge is inserted anaesthesia

deepened and the operation recommenced. Sometimes a second bout of vomiting follows after a short interval. After this has been dealt with in the same way the patient usually settles down peacefully and when he recovers consciousness may be quite unaware that he has vomited.

Advantage should be taken of any pause in the operation to make sure that the mouth is as dry as possible and free from any foreign bodies. The sponge should be replaced by a fresh one if during the course of an operation it becomes soaked in blood and mucus and no longer serves as an efficient protection.

### Co-operation between Dentist and Anæsthetist

Just as smoothness in induction is achieved by co-operation between patient and anæsthetist, maintenance of smooth anæsthesia and attainment of good operating conditions depend on good team work between dentist and anæsthetist. Unless both the dentist and anæsthetist have confidence in each other and work in harmony they are not likely to achieve much success. Any decision based on the depth of anæsthesia as to when the operation should be started, interrupted or terminated is one solely for the anæsthetist. In this he should never be questioned and his decision should be acted on at once. Many an anæsthesia has been ruined because the dentist has begun to extract before the anæsthetist wanted him to and even more because the dentist has failed to stop *immediately* in the course of an operation on request. The chief reasons for such a request are (i) That anæsthesia is too light and respiration shows signs of becoming oral. If the mouth is not covered up *at once* until nasal respiration is re-established the patient, particularly if resistant, will soon get out of control. (ii) That anæsthesia is too deep. (iii) That respiration is obstructed. The anæsthetist must not on these occasions be diffident about interrupting the operation even at a time inconvenient to the surgeon. The interests of the patient must come first and no other consideration can be allowed to outweigh this obvious fact.

The anæsthetist, besides providing smooth anæsthesia, should keep the patient's head in the best position for the dentist and should steady it throughout. He should keep the airway clear, steady the jaw, retract the tongue and keep the field of operation as clear as possible by mopping away blood with a sponge and so allow the identification of a root before forceps are applied. Lastly the anæsthetist should stand in such a position that he is out of the dentist's way and yet throughout can keep an eye on the patient's face or on the operation. The anæsthetist should relieve the dentist of as much responsibility and anxiety as possible. With a well-given dental anæsthetic there

need be no hurry at all and the dentist should always feel that he has ample time. He should be steadied down or encouraged as occasion arises. Advice such as 'Take it easy for a moment or two' or 'Have a minute's rest,' if given at the right moment, may be valuable. Experience shows that where a dentist takes a few minutes in loosening a tooth his arm becomes tired, his actions less expert, and in putting a little extra force on the tooth at this stage he may easily break it. If the anaesthetist thinks that the dentist is reaching this state it is best to suggest a thirty-second rest. The dentist returns refreshed and frequently accomplishes the extraction successfully. The anaesthetist should make it his duty to see that the patient's clothing is not soiled by blood and saliva dripping out of the mouth during a long operation. These sometimes drain along the tape attached to the sponge or mouth-pack and care should be taken to see that this does not project below the towel on the patient's chest. The anaesthetist often can prevent soiling of the clothing by folding the towel into such a position that it will mop up any blood or saliva which trickles down, or he may even find it necessary temporarily to suspend the operation, mop the mouth, particularly the sulcus between tongue and jaw and change the sponge.

### POSITIONING THE HEAD TO ALLOW ACCESS TO SPECIAL TEETH

The difficulty of extraction is increased in the case of certain teeth by their situation. The anaesthetist by appropriately manoeuvring the mandible can make the lower molar teeth and the upper third molar tooth more accessible to the dentist.

If the lower jaw is not supported the airway becomes obstructed. Apart from this it will be found difficult with the jaw in this position to apply forceps to the molar teeth particularly the lower right ones.



FIG 83

deepened and the operation recommenced. Sometimes a second bout of vomiting follows after a short interval. After this has been dealt with in the same way, the patient usually settles down peacefully and when he recovers consciousness may be quite unaware that he has vomited.

Advantage should be taken of any pause in the operation to make sure that the mouth is as dry as possible and free from any foreign bodies. The sponge should be replaced by a fresh one if during the course of an operation it becomes soaked in blood and mucus and no longer serves as an efficient protection.

### Co operation between Dentist and Anæsthetist

Just as smoothness in induction is achieved by co operation between patient and anæsthetist maintenance of smooth anæsthesia and attainment of good operating conditions depend on good team work between dentist and anæsthetist. Unless both the dentist and anæsthetist have confidence in each other and work in harmony they are not likely to achieve much success. Any decision based on the depth of anæsthesia as to when the operation should be started interrupted or terminated is one solely for the anæsthetist. In this he should never be questioned and his decision should be acted on at once. Many an anæsthesia has been ruined because the dentist has begun to extract before the anæsthetist wanted him to and even more because the dentist has failed to stop *immediately* in the course of an operation on request. The chief reasons for such a request are (i) That anæsthesia is too light and respiration shows signs of becoming oral. If the mouth is not covered up *at once* until nasal respiration is re established the patient particularly if resistant will soon get out of control. (ii) That anæsthesia is too deep. (iii) That respiration is obstructed. The anæsthetist must not on these occasions be diffident about interrupting the operation even at a time inconvenient to the surgeon. The interests of the patient must come first and no other consideration can be allowed to outweigh this obvious fact.

The anæsthetist besides providing smooth anæsthesia should keep the patient's head in the best position for the dentist and should steady it throughout. He should keep the airway clear steady the jaw, retract the tongue and keep the field of operation as clear as possible by mopping away blood with a sponge and so allow the identification of a root before forceps are applied. Lastly the anæsthetist should stand in such a position that he is out of the dentist's way and yet throughout can keep an eye on the patient's face or on the operation. The anæsthetist should relieve the dentist of as much responsibility and anxiety as possible. With a well given dental anæsthetic there

need be no hurry at all and the dentist should always feel that he has ample time. He should be steadied down or encouraged as occasion arises. Advice such as "Take it easy for a moment or two" or "Have a minute's rest," if given at the right moment, may be valuable. Experience shows that where a dentist takes a few minutes in loosening a tooth his arm becomes tired, his actions less expert, and in putting a little extra force on the tooth at this stage he may easily break it. If the anaesthetist thinks that the dentist is reaching this state it is best to suggest a thirty second rest, the dentist returns refreshed and frequently accomplishes the extraction successfully. The anaesthetist should make it his duty to see that the patient's clothing is not soiled by blood and saliva dripping out of the mouth during a long operation. These sometimes drain along the tape attached to the sponge or mouth-pack and care should be taken to see that this does not project below the towel on the patient's chest. The anaesthetist often can prevent soiling of the clothing by folding the towel into such a position that it will mop up any blood or saliva which trickles down or he may even find it necessary temporarily to suspend the operation, mop the mouth particularly the sulcus between tongue and jaw, and change the sponge.

### POSITIONING THE HEAD TO ALLOW ACCESS TO SPECIAL TEETH

The difficulty of extraction is increased in the case of certain teeth by their situation. The anaesthetist by appropriately manoeuvring the mandible can make the lower molar teeth and the upper third molar tooth more accessible to the dentist.

If the lower jaw is not supported the airway becomes obstructed. Apart from this it will be found difficult with the jaw in this position to apply forceps to the molar teeth particularly the lower right ones.



FIG. 83

## Extraction of Lower Teeth



FIG 84

For the extractions of all lower teeth the mandible must be steadied and pushed forward by pressure behind the angle. The farther back the tooth the more will this slight dislocation forward of the mandible assist the application of forceps.

The nose piece is steadied by the thumb and thenar eminence whilst the ring finger feels the pulsations in the external carotid artery.

## Extraction of Lower Molars

When molar teeth are to be extracted additional help is afforded if the anæsthetist turns the chin in the opposite direction and raises it slightly.

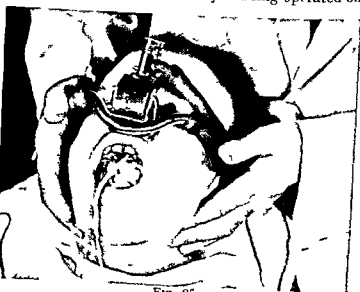


FIG 85

When a left lower molar is to be extracted the mandible is pushed well forward the head is allowed to fall slightly to the left the chin turned to the right and slightly raised.

For extractions of left lower molars the dentist usually stands in front of the patient but when extracting right lower molars he generally stands behind

When a right lower molar is to be extracted the anaesthetist remains on the left side of the patient but moves forward slightly. His right arm still encircles the patient's neck and the jaw is steadied and pushed forward by both hands. In this position the anaesthetist still has a clear view of the patient's face. The head is allowed to fall to the right the chin turned to the left and slightly raised



FIG 86

### Extraction of Upper Teeth

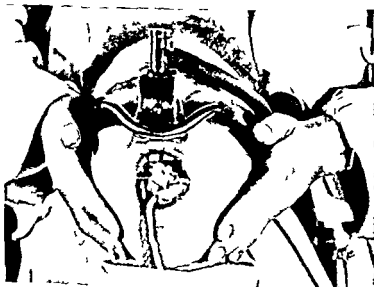


FIG 87

The head must be steadied between the palms of the hands and counter-pressure may be applied by the anaesthetist's chest pressed on the top of the head



## Extraction of Upper Third Molar Teeth

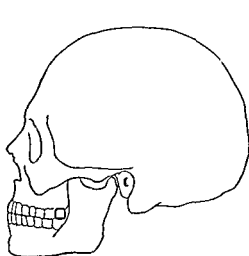


FIG 88

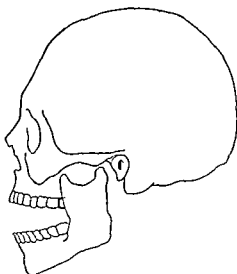


FIG 89

When the mouth is opened the head of the condyle of the mandible slips forward on the eminentia articularis and the coronoid process of the ascending ramus of the mandible is brought into close proximity to the upper third molar tooth. The proximity of the coronoid process makes it difficult for the dentist to apply his forceps to the upper third molar tooth.



FIG 90



FIG 91



FIG 92

Access is much improved if the anæsthetist pushes the mandible towards the side of the extractions thus displacing the coronoid process.

If the upper third molar on the left side has to be extracted the anæsthetist with his right hand pushes the mandible to the left thus

carrying the left ascending ramus away from the maxilla sufficiently to allow forceps to be applied to the tooth

In extracting upper incisor teeth, the dentist sometimes finds the nose-piece is in his way if he wishes to get his fingers on the alveolar process under the nose. If the nose-piece is pushed upwards by the dentist's manipulations gas must be delivered from it under marked positive pressure to prevent the leakage in of air which would otherwise occur



FIG 93



FIG 94

During the extraction of teeth from the upper jaw the lower lip can be pinched easily between the lower teeth and the forceps. We have seen the forceps pressing so heavily that a lower tooth has cut completely through the lip. If by any chance the lower lip is bruised or perforated in this way since the tissues in this region are very lax a big hæmatoma can result. If pressure is applied quickly the production of a hæmatoma is prevented and because of the vascularity of the part healing in this region is extremely rapid. The anæsthetist can prevent this accident by hooking his finger inside the lower lip and pulling it away as shown in fig 94

## RECOVERY FROM NITROUS-OXIDE ANÆSTHESIA

When extractions are completed the patient should not be disturbed but should be allowed to recover consciousness in his own good time. He may pass into natural sleep and then should not be roused for a few minutes. During the waking period patients are frequently capable of appreciating what is going on around them and realise that by exerting themselves a little they would become fully awake but they are reluctant to make the effort and continue to sit quite still enjoying a comfortable lethargy. Hearing the last sense to disappear during induction is the first to return. Caution should therefore be exercised in any conversation which takes place while the patient is recovering consciousness and any observations should be encouraging e.g. 'That was very successful,' or 'He was a very good patient.'

The prop should not be removed from the mouth until the patient is fully conscious. It is alleged that if this is done the stimulus may be interpreted as that of extraction of a tooth. We do not believe this confusion ever exists but the advantage of leaving the prop in the mouth is that it prevents swallowing. The sponge also should be left in the mouth till the patient is conscious enough to spit it out himself. By absorbing blood and saliva, it prevents these from being swallowed or from running down the side of the mouth. The patient should not be encouraged to spit out until he is fully orientated or he may do so over the floor. Similarly there is a danger of his reflexly drinking the mouth-wash if this is held to his lips before he is fully conscious.

### Subjective Sensations on Recovery

Patients' sensations on recovery show much variation. After nitrous-oxide anæsthesia lasting say 3 minutes a patient by his remarks may reveal for how long he feels that he has been unconscious. His idea of the duration of the anæsthesia enables a fairly accurate estimate to be made of his nervous or mental stability at the time of the operation. The very healthy patient passes off the experience as though nothing had happened—for example he will make no comment at all or if interrogated will say he has been asleep for only a few seconds—but the unstable individual is a long time before regaining a sense of normality and may say he feels that he has been away for an hour or two or longer or even that he has been away for years and has just come back to earth again. In extreme cases patients suffer in varying degrees from a sense of unreality for some hours.

Since for some people the state of anaesthesia has a mental association with death remarks to the recovering patient such as "Now you are coming back to earth" or "It is all over" are unwise. Especially after long  $N_2O$  anaesthesia an introspective patient may for some time find it difficult to orientate himself and remarks such as these do not help him. A gentle pat on the back and a cheerful "Well done" encourage quick recovery. In general the patient should not be told of it if he has been a difficult subject for anaesthesia, because the knowledge may make him apprehensive on a future occasion. If however he has struggled so violently as to be a danger to himself then in his own interests he should be told, so that another time he can warn the anaesthetist of his previous behaviour.

### Dreams

Recovery from general anaesthesia is usually a slow process unaccompanied by vivid dreams. In the case of  $N_2O$  in the dental surgery however recovery is abrupt and the patient frequently experiences a vivid dream, the outline of which is remembered but of which the details rapidly fade as the attempt to relate it is made. The dream may contain matter of vital importance such as the key to the solution of the riddle of the universe may cause dejection and terror or it may provoke no discernible emotional reaction. Dreams which are definitely funny are as rare as they are among dreams occurring during waking from natural sleep. Distressing dreams and unpleasantly vivid nightmares sometimes upset a patient sufficiently to make him dread inhalation anaesthesia from a well justified fear of a repetition of the experience. This dread is often the greater because the patient cannot remember quite what frightened him however real the dream seemed at the time of awakening. An adult who has had such a dream is as difficult to reassure as a child after a night terror and usually finds himself equally unable to describe his experience.

The dream may be associated with something in the patient's mind before the anaesthetic is commenced. For example it is common for a few days preceding the Derby for the patient to wake up witnessing with the greatest clearness the *finish* of the race. Within a few seconds however the previously easily recognisable racing colours become drab and the more fully the patient recovers the more to his bewilderment and disappointment do the details of the dream disappear into the background.

A dream or nightmare resulting from inadequate anaesthesia is not uncommon. During very light anaesthesia a stimulus may not be felt

as pain but may give rise to a nightmare Two cases on the same morning in the out patient department illustrate this point

A robust man was lightly anæsthetised for an extraction of a lower molar On recovery in answer to a question he said that his dream was that he had been boxing Jack Dempsey Questioning brought out the fact that in his dream he had been knocked out by a blow on his jaw and further that the blow had been delivered at the point from which the tooth had been extracted

A lightly anæsthetised woman had several upper teeth extracted She answered on recovery that she dreamt that she was being pulled through a tunnel by her hair

As in dreams on awakening from natural sleep the action in dreams on recovery from  $N_2O$  may seem to the patient to have covered several hours though the whole period of anæsthesia may have occupied only a minute Indeed dreams on emergence from sleep or anæsthesia often take place in a fraction of a second That this is so is shown by the common experience that some stimulus which rouses the sleeper appears as the final event in the dream for example a knock on the door initiates a dream in which the dreamer takes a long journey with many adventures and finally reaches a house where he knocks for admittance He wakes to find himself in bed and realises that he has been roused by the knocking on the door A drop of rain on the face of a person asleep in the open although it appeared to wake him instantly was accompanied by a dream the action of which lasted for some minutes In this an outbreak of fire was witnessed the summoning and journey of the fire brigade and the connecting of the fire hoses to the hydrants all took time and finally the spray of the hose which was to have been directed on the burning house hit the dreamer who awoke to feel the drop of rain

As far as possible, external stimuli therefore should be eliminated during the time when the patient is recovering consciousness, since they may initiate dreaming During the course of dental anæsthesia it is easy for a woman's skirt to become disarranged by her slipping down in the chair or moving her legs If this is not noticed until the patient is recovering it is advisable to let the patient recover and adjust her skirt herself It is possible in the rare cases where a patient alleges that misconduct occurred while she was unconscious that such a readjustment of the skirt was the stimulus leading to an erotic dream retrospective and ending with the pulling down of the skirt This last part of the dream really took place but the part preceding it might seem

equally real and credible to an excitable patient. Even without a definite stimulus erotic dreams are known to occur during recovery from N<sub>2</sub>O anaesthesia and we repeat here that to safeguard himself against charges of misconduct a doctor or dentist should never administer an anaesthetic unless a third person is present.

### Post-operative Nausea and Vomiting

Vomiting after the recovery of consciousness is disappointing and distressing for the patient and in the dental surgery it is an annoyance to the dentist because it lengthens the period for which a patient must be kept in the surgery.

Various factors which increase the incidence of vomiting include

Personal idiosyncrasy of the patient Good sailors and patients who tolerate a fair amount of alcohol come into the category of those undisturbed by anaesthesia. The patient who is a 'poor traveller' and who readily becomes nauseated in trains or on a ship is likely to suffer from post anaesthetic malaise. That temperament is a more important factor in the causation of vomiting than is the presence of food in the stomach is illustrated by the behaviour of the placid individual who has a normal meal within an hour of the time fixed for dental extraction. The fact that he has so little apprehension that he can enjoy a good meal in these circumstances is almost an assurance that he will not suffer from nausea after a short anaesthetic with nitrous oxide.

Type of anaesthetic used Nitrous oxide and vinylene seldom cause vomiting in ordinary dental extractions but ethyl chloride is not infrequently an offender. Thiopentone and other barbiturates seldom cause vomiting but in rare cases their use has been followed by nausea and vomiting lasting up to twenty hours during which time the patient may be prostrate and suffer from giddiness.

Depth and length of anaesthesia

Prolonged anoxia

Swinging anaesthesia i.e. maintenance of an uneven level of anaesthesia

Administration of gases under pressure The distension of the lungs by gases under pressure is followed by sweating collapse and nausea as is distension of any other hollow viscus.

Fasting Prolonged abstinence from food in order to avoid vomiting generally defeats its own end. Even short anaesthesia and slight deprivation of oxygen in a starved patient particularly one not robust is followed by a fall in blood pressure nausea and possibly retching.

Vomiting is often wrongly attributed to the swallowing of blood. If a patient vomits after an operation on the throat or mouth blood will inevitably be brought up and may then be held responsible for the vomiting. It is however only necessary to consider the high percentage of cases in which the swallowing of blood is not followed by vomiting to realise the unimportance of this factor. Further when vomiting occurs after operations on any other parts of the body blood is not vomited because none has been swallowed. If the operation had been on the nose or mouth the swallowed blood might have been given as the cause of the vomiting whereas the presence of blood in the vomitus was only coincidental.



FIG 95

The treatment of post operative nausea by means other than postural are disappointing.

The patient's head should be pushed well down between his knees and he should be told to cough gently once or twice. These procedures raise the blood pressure and are usually followed by flushing of the face and by considerable though often only a temporary general improvement and if the patient sits upright again he relapses. Stimulation by smelling salts may be effective. The small capsules of aromatic ammonia are the most convenient and reliable when compressed ammonia vapour is liberated. Whether brandy or

anything else should be given by mouth at this stage is an open question. Such treatment usually augments a feeling of nausea and actually precipitates vomiting but the act of vomiting then often relieves the nausea.

If these restorative measures are not effective the patient should be allowed to rest in a recumbent position. Fifteen minutes rest immediately after the operation is of more value than an hour's rest later on and the dentist who operates in his surgery on cases requiring prolonged anæsthesia should have a small recovery room. The patient's head should be kept low and he should be covered with a blanket to keep him warm and comfortable. It is advisable to have a bowl and

a towel at hand in case of vomiting. If a patient is to be driven home before he feels that he has recovered completely it is a thoughtful precaution to give him a bowl and a towel since a feeling of nausea in the surgery is not infrequently intensified once the patient is in a moving car, and may be followed by vomiting.

A patient should be strongly persuaded not to drive his car himself soon after recovering from an anæsthetic. Patients often stubbornly insist on driving home but so many subsequently confess that they drove 'as if in a dream' that it is strange that accidents in such circumstances appear to be so extremely rare.

### REFERENCES

- (1) Clement F W 1939 *Nitrous Oxide Oxygen Anesthesia* London 136
- (2) Macintosh R R and Pratt Freda B 1938 Contribution (Chapter 3) to *Modern Anæsthetic Practice* London 71
- (3) Macintosh R R 1931 *Brit dent J* 52 (1) 89
- (4) Paterson H J 1899 *W Lond med J* 4, 202



## CHAPTER XXV

## ENDOTRACHEAL ANÆSTHESIA

FOR many operations endotracheal anæsthesia is not merely a refinement but an essential. The anæsthetist therefore must be proficient in the art of intubation.

For example, if general anæsthesia is to be used for any <sup>①</sup>prolonged operation on the head and neck, it should be administered through an endotracheal tube so that the anæsthetist can keep away from the field of operation and yet retain full control of the airway. In addition, for operations in which blood, teeth, or other foreign bodies may be free in the mouth, this technique allows the laryngeal entrance to be packed off so giving complete protection against their entry to the air passages.  
 ③ For abdominal surgery too, endotracheal anæsthesia is valuable in that it obviates the possibility of laryngeal spasm as a response to certain intra abdominal manipulations, particularly traction on the stomach or gall bladder.

The use of a wide bore tube ensures a free airway and thus eliminates the main cause of difficulties and disasters during anæsthesia. Also the depth of anæsthesia can be readily controlled and the easy expansion of the lungs with every breath minimises the chances of occurrence of post operative atelectasis and the serious conditions which may follow. The complications attributed to passing an endotracheal tube have been greatly magnified and are almost invariably accounted for by faulty technique.

The tube may be passed through the mouth or through the nose. When the oral route is chosen, the method almost invariably adopted is to expose the larynx with a laryngoscope and to pass the tube under direct vision. When the tube is passed through the nose a similar procedure may be followed or the tube may be made to enter the larynx blindly.

Blind intubation carries with it many advantages but not the assurance that the tube can be passed through the nose into the trachea in every case. Direct-vision laryngoscopy may be slightly more difficult but proficiently performed ensures the passage of an endotracheal tube. On account of its certainty this method should be mastered early since it is to this that the anæsthetist invariably has recourse when blind intubation fails.

In many cases 'blind' intubation is strikingly easy. In general it is a simple procedure on men, but is frequently more difficult in the edentulous and in narrow faced women and in children

There is no doubt that with practice a high percentage of success can be achieved but the practitioner must not overlook the virtue of knowing how to manipulate a laryngoscope skilfully. There always remain a few cases which cannot be intubated blindly and in these it is a comfort to be able to pass the tube neatly under direct vision.

Direct-vision intubation is regarded by some as a difficult procedure, but this is mainly because the underlying principles are not understood. These are straightforward and if properly appreciated and their implications brought into practice it becomes a simple manœuvre.

Relaxation of the jaw and abolition of the laryngeal reflex are essential. Also the neck and head must be correctly placed—the neck must be flexed and the head extended on the atlanto occipital joint. This

Chevalier Jackson position <sup>1,2</sup> is easily achieved by keeping the shoulders on the table, placing an extra pillow under the occiput—and then extending the head.

## MECHANICS OF LARYNGOSCOPY

The following series of diagrams and X ray records shows the anatomical essentials

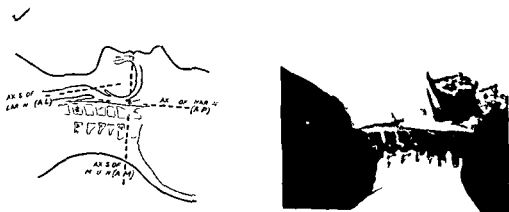


FIG. 96.—Relative positions of the axes of mouth, pharynx and larynx with patient lying flat.

The axis of the mouth forms approximately a right angle with that of the pharynx and when the patient is lying flat the latter crosses the axis of the larynx and trachea.

The main mechanical problem is to bring these three axes into one

Fig 97 illustrates the anatomical effect achieved by inserting a high pillow beneath the occiput thus flexing the neck. The pharyngeal and laryngeal axes are thus made to coincide

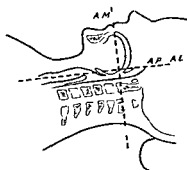


FIG 97 —Showing how axes of pharynx and larynx may be made to coincide when the head is raised. The cervical spine is also straightened out

As Gillespie has pointed out <sup>4</sup> in order now to straighten out the right angle formed by the axis of the mouth and the pharyngolaryngeal axis, the head must be extended about the atlanto occipital joint

The head is fully extended. The laryngoscope lifts the patient's lower jaw and epiglottis. It must not take leverage off the upper teeth

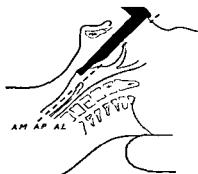


FIG 98 —Axis of the mouth is now made to coincide with the other axes by extension of the head around the atlanto-occipital and upper cervical joints

though it almost inevitably touches them. As Jackson <sup>1</sup> has suggested the patient's head is suspended on the base of his tongue and epiglottis. Further when the head and neck are in this position the epiglottis is viewed through the laryngoscope almost end on and is very easily passed by the blade of the instrument. Indeed the laryngoscope is often

passed to the larynx without the epiglottis being seen at all, much less getting in the way

This position is essential for the passage of a bronchoscope. The requirements of bronchoscopist and anæsthetist differ only in that the former passes a rigid tube, and so must have complete accuracy of exposure while the latter uses a soft or semi rigid tube, the flexibility of which enables him to overcome inaccuracies of technique. The anæsthetist who places the head of his patient in the position illustrated will be agreeably surprised to find how much easier his task becomes.

### DIRECT-VISION LARYNGOSCOPY

Laryngoscopy should first be practised in the post mortem room. Later the anæsthetist should expose the larynx in the patient still deep under anæsthesia *at the end* of an operation. The patient is then completely relaxed and the anæsthetist has plenty of time to familiarise himself with the appearance of the larynx under conditions ideal for intubation.

If the tube is to be passed through the nose five to ten minutes before passing the tube the vascularity of the mucous membrane of the nose should be diminished by spraying it with cocaine. For this purpose a 10 per cent or 20 per cent solution is recommended and it is often suggested though in our opinion unconvincingly that owing to the intense vasoconstriction produced by strong solutions less cocaine is absorbed if the 20 per cent rather than the weaker solution is used. The spraying can be done when the patient is conscious but since the intensely bitter taste of cocaine is disagreeable it is considerate to induce anæsthesia first.

The larynx also can be cocaineised if desired by inserting the nozzle of the spray into the nostril and spraying during inspiration. If the larynx and trachea are not cocaineised third-plane anæsthesia is necessary to depress the reflexes sufficiently for the tube to be tolerated. If however cocaine is used the tube will be tolerated even during upper first plane anæsthesia for example under nitrous oxide alone.

The distal half or two thirds of an endotracheal tube should always be lubricated before being passed. This facilitates the passage of the tube through the nose and, especially when a large tube is used, reduces the risk of damaging the turbinate bones. Vaseline may be used as the lubricant but an ointment containing a local anæsthetic for example, 10 per cent nupercaine ointment may be advantageous where it is desirable to prolong the effect of the cocaine previously sprayed on the cords thus keeping the laryngeal reflexes depressed.

The length of the tube used is important. As supplied by the

makers all tubes are intentionally longer than required so that the anæsthetist may cut them to the desired length. Unless it is shortened there is a real danger that the tube will enter one main bronchus usually the right. There is then the danger of the unaerated lung collapsing (p 267). The length of a tube should be 1½–2 times the distance from the nostril to the lobe of the ear

Under general anæsthesia the mobility of the jaw is a good criterion of adequate relaxation and until the masseter is fully relaxed the laryngoscope must not be introduced. If because of a poor airway it is difficult to deepen anæsthesia sufficiently to relax the jaw a short naso-pharyngeal tube may be introduced after which the intake of anæsthetic vapour will be greatly improved. Such a tube is more readily tolerated in light anæsthesia than is an oral airway which requires almost as much relaxation of the jaw for its introduction as does the

laryngoscope itself

The head is now placed in the correct position either by placing the occiput on the head rest shown in fig 101 by slipping a second pillow under the head or by folding double the one already present

The anæsthetic mask is lifted from the face and the nasopharyngeal tube (if used) removed. The endotracheal tube is passed directly backwards into the nostril but gently and if any obstruction is encountered it should be withdrawn and passed through the other. Force must never be used in passing the tube since the soft tissues are easily damaged. After the tube has been passed 2–3 inches some resistance may be encountered from the sphincter like action of the upper end of the superior constrictor muscle of the

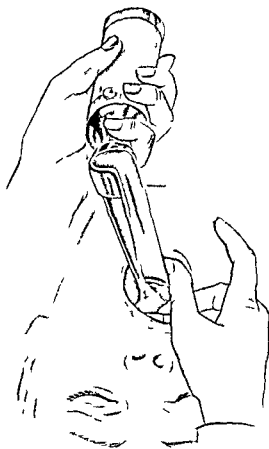


FIG 99

pharynx. After this has been overcome by gentle pressure the patient will be heard breathing through the tube. The tip of the tube now lies in the pharynx ready to be inserted into the larynx when this has been exposed.

The chin is now tilted sharply upwards, so as to extend the head about the atlanto occipital joint. The laryngoscope is held in the left hand throughout leaving the right hand free. While the blade is being introduced the lips are drawn back to prevent them from being nipped between teeth and blade.

We find it convenient to rest the second finger of the right hand on the upper teeth and to open the mouth by depressing the lower jaw by the thumb (fig 99).

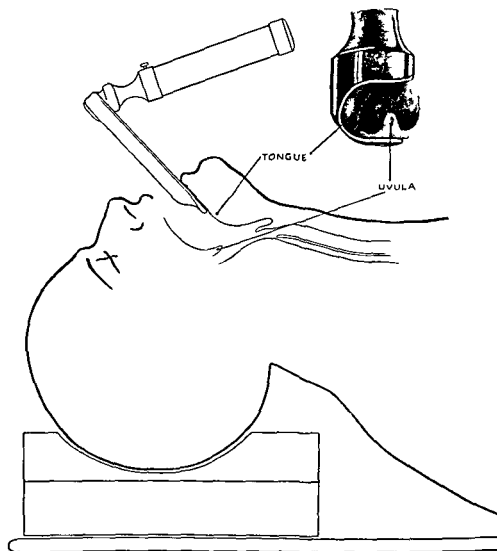
The blade is inserted well to the right side of the mouth and as it is advanced the tongue is pushed towards the left. The avoidance of the midline anteriorly shortens the distance from teeth to larynx and this makes the introduction of the laryngoscope easier. It also minimises the risk of damaging the teeth because the laryngoscope blade is in contact with the molar not the more fragile incisor teeth. Further it prevents the tongue from bulging over the lateral cleft of the laryngoscope obscuring the view of the larynx.

Some anaesthetists advise that the upper front teeth should be covered with a thin sheet of lead or with sticking plaster to prevent them from being chipped by the blade of the laryngoscope. This accident should not occur however if the laryngoscope is properly used.

The object of this diagram (fig 100) is to warn against using the blade in the midline and as a lever with the upper teeth as a fulcrum with probable chipping or dislodgment of these teeth.



FIG 100

FIG 101 (After Thomas<sup>5</sup>)

When the laryngoscope has advanced about half the distance it must eventually travel, the uvula comes into view

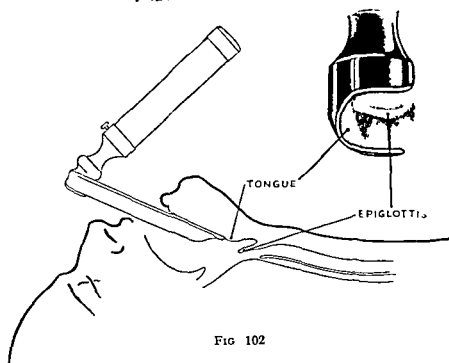


FIG 102

When it has reached to two thirds of the full distance, the tip of the laryngoscope is directed towards the midline and raised until the epiglottis is seen

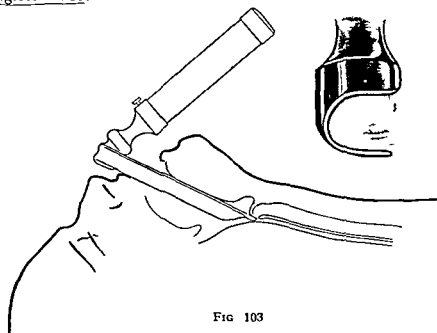


FIG 103

Still advancing the tip of the laryngoscope is directed towards the posterior pharyngeal wall and passed to about half an inch beyond the epiglottis



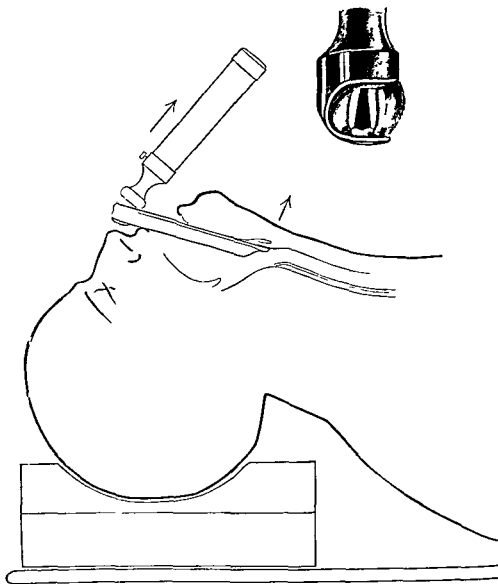


FIG 104

The handle of the laryngoscope is now lifted so that the blade exerts a steady pressure equally throughout its whole length, on the tissues with which it is in contact. The whole head is now lifted on the blade until the vocal cords come into view.

Once the larynx is exposed the tip of the tube can generally be guided with ease through the glottis simply by manipulating the end which projects from the nose

Should manipulation of the proximal end of the tube be unsuccessful the distal end is picked up with a pair of Magill's forceps and guided into the larynx

When oral intubation is practised a very flexible tube is unsuitable for it is liable to kink or to get compressed. This does not apply however to the flexible armoured tubes designed to avoid these dangers. To facilitate intubation a knitting-needle may be passed through the tube to straighten it and to make it rigid. As soon as the tube is in position and the knitting needle has been withdrawn a Rowbotham metal connection is

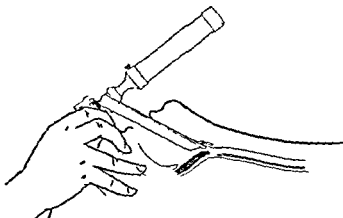


FIG 105

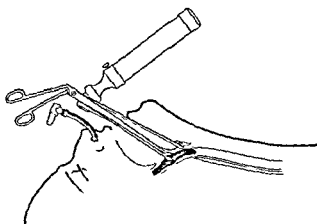


FIG 106

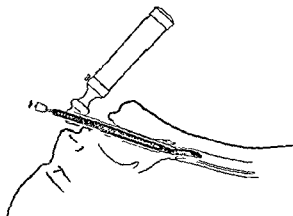


FIG 107

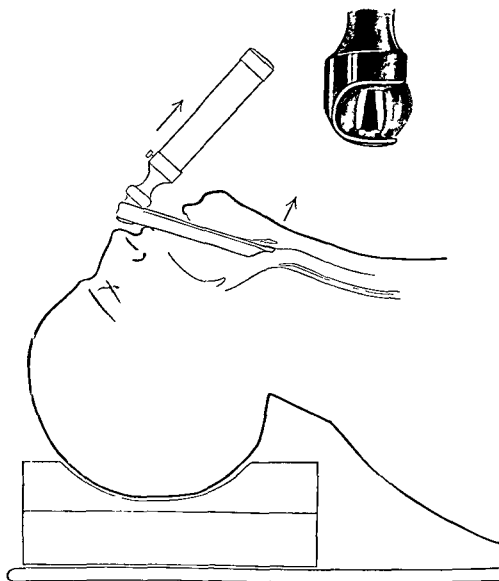


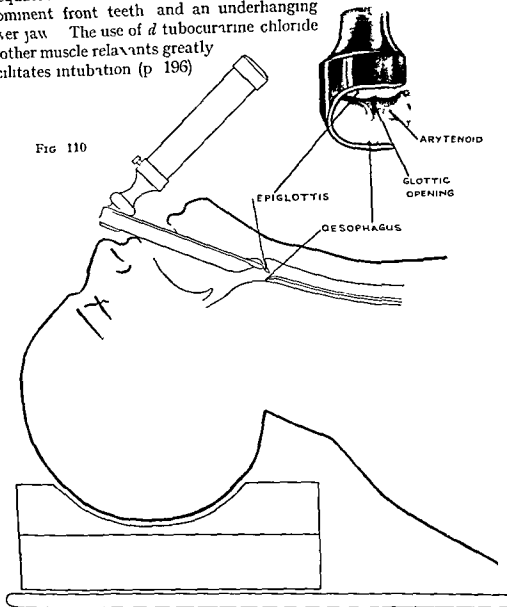
FIG 104

The handle of the laryngoscope is now lifted so that the blade exerts a steady pressure equally throughout its whole length, on the tissues with which it is in contact. The whole head is now lifted on the blade until the vocal cords come into view.

## CAUSES OF DIFFICULTY IN EXPOSING THE CORDS

The main cause of difficulty is lack of experience together with failure to recognise landmarks particularly when the laryngeal muscles are not relaxed. Contributory causes of difficulty are inadequate anaesthesia, failure to flex the neck when the head is extended, prominent front teeth and an underhanging lower jaw. The use of *d* tubocurarine chloride or other muscle relaxants greatly facilitates intubation (p. 196).

FIG 110



There may be difficulty in hooking the epiglottis when this is folded backwards against the arytenoids. To move it so that the tip of the blade of the laryngoscope can be passed behind it the anaesthetist should push the thyroid cartilage upwards with the fingers of his right hand.

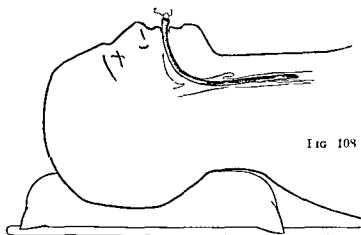


FIG 108

attached to the proximal end

If an airtight junction between the anæsthetic circuit and the patient's lungs is advisable to prevent inhalation of blood during operations on the mouth or nose or for closed

circuit anæsthesia one way of accomplishing this is to use a tube surrounded with an inflatable cuff. When the tube is in position the cuff is inflated through its pilot tube with air from a syringe. The pilot tube is clamped by a paper clip when the degree of distension of the small balloon at the proximal end indicates that the tension in the cuff is adequate.

Alternatively, an airtight junction can be achieved by inserting a pack round the tube at the laryngeal entrance.

c) Two inch gauze bandage is satisfactory. This should be lubricated to prevent abrasion of the mucous membrane of the pharynx by the gauze. The bandage is soaked in liquid paraffin and autoclaved on a wire mesh tray. This sterilises the pack and allows excess paraffin to drain off so that paraffin will not leak alongside the tube into the lungs. The gauze is packed firmly but gently round the tube either with the fingers or under direct vision using Magill forceps. Several inches of bandage should be left hanging out of the mouth and the anæsthetist should make himself responsible for the removal of the pack before the patient leaves the operating theatre.

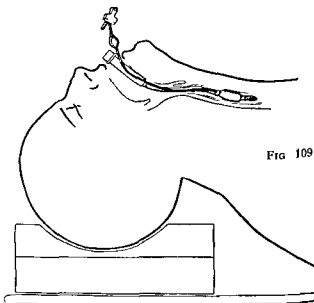
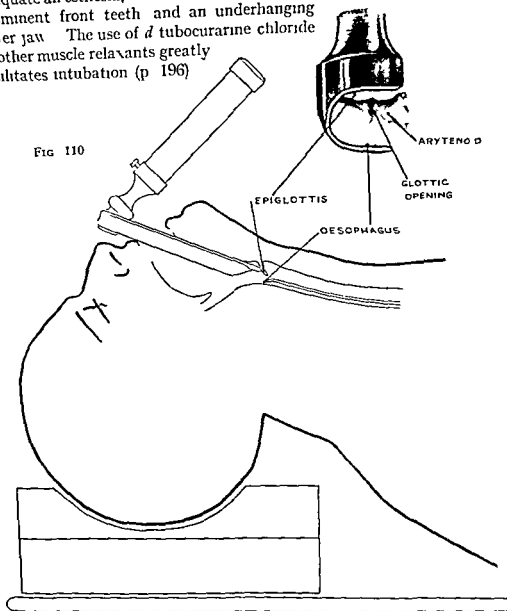


FIG 109

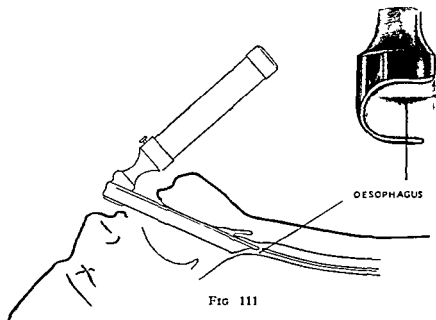
## CAUSES OF DIFFICULTY IN EXPOSING THE CORDS

The main cause of difficulty is lack of experience together with failure to recognise landmarks particularly when the laryngeal muscles are not relaxed. Contributory causes of difficulty are inadequate anaesthesia, failure to flex the neck when the head is extended, prominent front teeth and an underhanging lower jaw. The use of *d* tubocurarine chloride or other muscle relaxants greatly facilitates intubation (p 196)

FIG 110

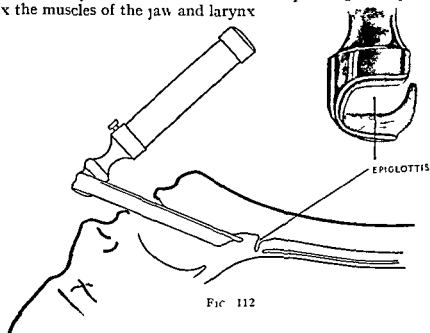


There may be difficulty in hooking the epiglottis when this is folded backwards against the arytenoids. To move it so that the tip of the blade of the laryngoscope can be passed behind it the anaesthetist should push the thyroid cartilage upwards with the fingers of his right hand.



If the blade of the laryngoscope is introduced too far the œsophageal opening comes into view

The following additional difficulties are all caused by attempts to expose the larynx before anæsthesia is deep enough adequately to relax the muscles of the jaw and larynx



During very light anæsthesia it is impossible to push the laryngo scope behind the epiglottis because this is folded backwards as the larynx is raised in the act of swallowing

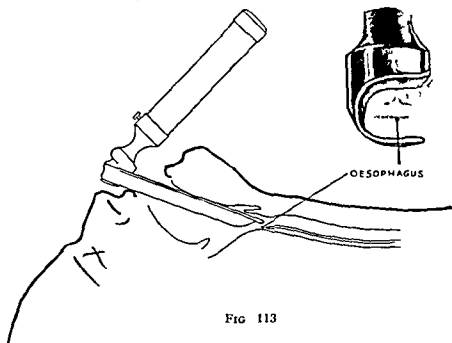


FIG 113

At a deeper level of anæsthesia the laryngoscope can be passed behind the epiglottis but since the muscles of the jaw are not relaxed they resist the lifting action of the laryngoscope. Complete exposure of the larynx is then impossible and since its muscles are in spasm only the approximated arytenoid cartilages come into view. Posteriorly the œsophageal opening is seen.

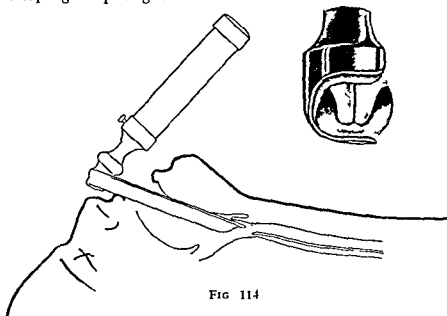


FIG 114

At slightly deeper levels of anæsthesia the jaw can be lifted enough to expose increasing amounts of the still approximated aryepiglottic folds.



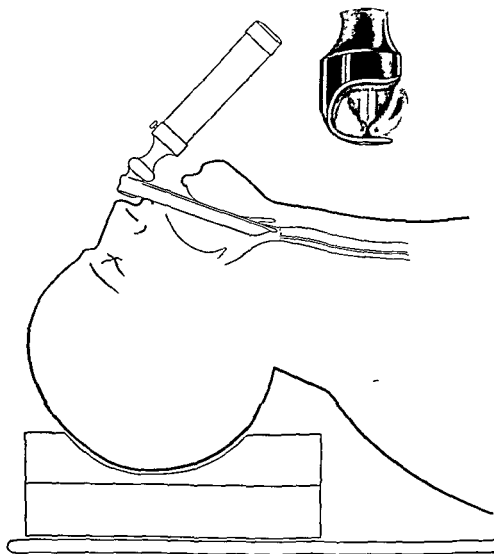


FIG 115

At a still deeper level of anæsthesia the muscles of the jaw relax and allow the laryngoscope to lift the lower jaw and expose the larynx more fully. The muscles of the aryepiglottic folds have also relaxed though the cords are still in spasm.

### “BLIND” NASAL INTUBATION

In directing the tube through the nose into the trachea the anæsthetist is guided by touch and sound. The main advantage of this procedure is that it can be carried out under light anæsthesia whereas

if the tube is passed under direct vision, the anaesthesia may have to be deeper than is required for the surgical operation itself. Before passing the tube through the nose the nose and larynx are cocaineised as described previously (p 249).

The same position as for direct vision laryngoscopy is used, sniffing the air position. The tube is introduced as described (p 250) and advanced until the breathing is heard through it. The anaesthetist now keeps his ear to the tube, and gently pushes it farther until

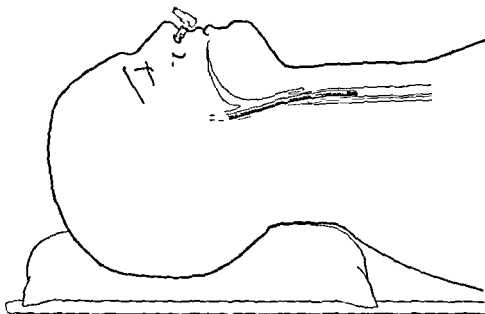


FIG 116

it either enters the trachea (fig 116), or enters the oesophagus (fig 117), or is held up at the laryngeal entrance (fig 119) or passes anterior to the epiglottis and lodges against the base of the tongue (fig 121).

The signs that the tube has entered the trachea (fig 116) are so obvious that when there is any doubt about it, it may be assumed that it has not done so. Unless the protective reflexes of larynx and trachea are much depressed either by cocaineisation or deep general anaesthesia its introduction into the trachea will lead to a paroxysm of coughing while if anaesthesia of these parts is complete the regular and easy respiration characteristic of surgical anaesthesia will be heard through the tube as soon as this has entered the trachea.

With the tube in the position shown in fig 117 the signs though negative are obvious. The tube has been inserted to its full length

and though respiration continues unimpeded breath sounds are not transmitted through it

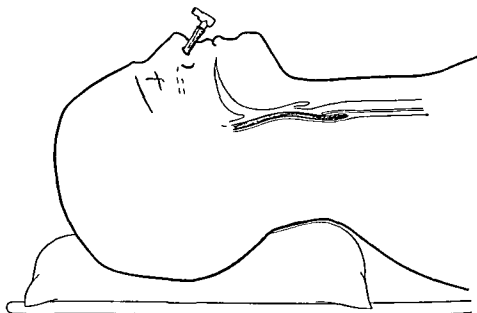


FIG 117

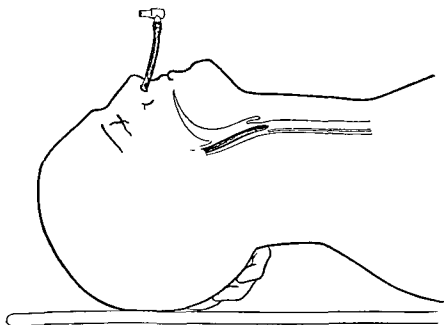


FIG 118

To correct this the tube is withdrawn slightly and its tip directed towards the larynx by extending the neck and head

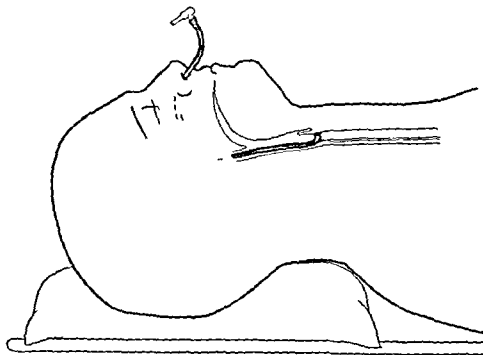


FIG 119

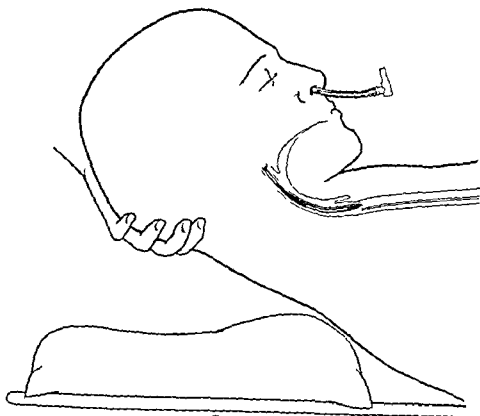
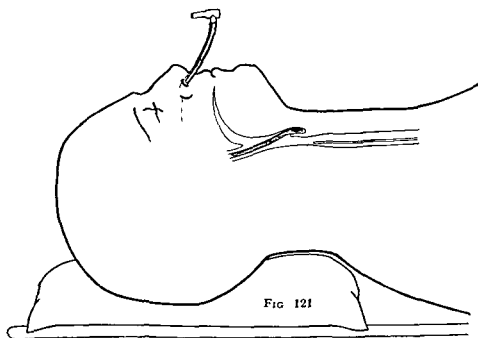


FIG 120

The tube may be arrested by the cords (fig 119) if these are fully or partially adducted a condition often found if the larynx is stimulated when the respiratory centre is depressed (e g by morphia) The breath may be held for some seconds or respiratory movement may be much diminished so that only faint yet characteristic croaking sounds are transmitted through the tube The distal end of the tube runs almost parallel to the cords and impinges on the posterior aspect of the epiglottis Pressure exerted on the tube in the hope that it will pass through the adducted cords kinks it while still further pressure may displace it laterally

A tube which is held up at the cords usually can be made to pass into the glottis. The head is flexed acutely (fig 120) and the distal end of the tube thereby brought almost at right-angles to the glottic opening a position which greatly facilitates its entrance By maintaining gentle pressure on the tube an attempt is made to insinuate its bevelled end between the cords If this fails the tube is moved slightly to irritate the laryngeal mucosa and stimulate coughing, when the cords will separate widely



This is the fourth position in which the tube may be arrested

To correct this malposition of the tube it must be withdrawn slightly and the head well flexed so that the tip of the tube is directed backwards behind the epiglottis (fig 122)

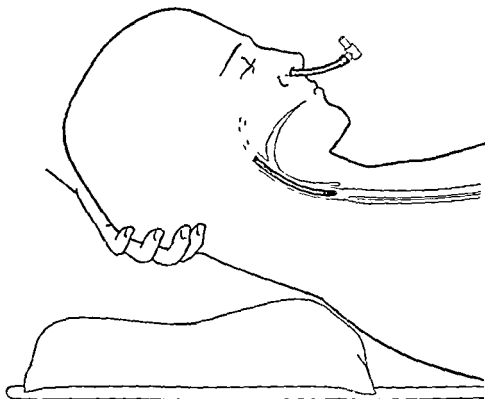


FIG 122

If for any reason difficulty is experienced in passing the tube blindly any of the following manœuvres may be of assistance the tube is withdrawn and passed through the other nostril or a tube with a different curve is chosen or respiration may be stimulated by giving  $\text{CO}_2$  to facilitate entrance into the larynx

### INTUBATION UNDER LOCAL ANÆSTHESIA

Laryngoscopy under local anæsthesia is not so formidable a measure as might be supposed. The throat is sprayed with 10 per cent cocaine and the right side of the tongue and, in an edentulous patient the gums, are painted with the solution applied on a cotton wool swab. After a pause a small cotton wool swab firmly secured in a curved applicator dipped in cocaine solution and subsequently gently squeezed to express excess cocaine, is held against the lateral wall of the pharynx just below the pillars of the fauces. It is gently advanced down the lateral wall of the pharynx until it is held up in the pyriform fossa. The process is repeated on the other side of the pharynx. Finally a third swab is passed until it comes to rest upon the vocal cords as

shown by the alteration in the sound of the breathing. This last swab should not be squeezed but left saturated with solution so that a few drops trickle into the upper part of the trachea. Provided the head and neck are placed in the correct position laryngoscopy and intubation should now be easy.

**1 Fractured jaw** — *Endotracheal anæsthesia is essential. If the lower jaw is shattered, and anatomical landmarks are altered, great difficulty may be experienced with general anæsthesia and intubation. Blood may have been inhaled and bleeding may not have stopped when the patient reaches the operating theatre. The tongue, lacking the retention of an intact lower jaw, falls back if the patient is in the supine position, and obstructs the laryngeal entrance. Many lives have been lost through ignorance of the simple mechanics involved and a patient with a fractured jaw should be kept in the semi prone position until he reaches the operating table. An additional safety measure is the insertion of a stitch in the tongue by means of which it may be pulled forwards.* This should be done when the patient is first seen.

Where there is much blood and debris, it is advisable to pass an endotracheal tube under direct vision using local anæsthesia alone. Once a clear airway has been established general anæsthesia can be induced with thiopentone and maintained either with thiopentone or by inhalation anæsthesia.

**2 Lung toilet** — Intubation under local anæsthesia is valuable for lung toilet in cases of post operative atelectasis where it is desired to apply suction to remove secretions. The nose is sprayed with cocaine and the pharynx and vocal cords cocaineised as above though not necessarily as thoroughly. With the conscious patient in the correct position the tube is usually passed blindly with ease.

## WARNINGS ON THE USE OF ENDOTRACHEAL ANÆSTHESIA

The fact that a tube has been passed into the trachea must not of itself be regarded as a guarantee that the airway is now assured. Before pronouncing the case ready for operation the anæsthetist must satisfy himself that there are no signs of respiratory obstruction or if there are any he must investigate and rectify the cause.

A tube which is too small, especially when the throat is picked off is equivalent to a persistent respiratory obstruction—it is true that air passes in and out of it but free respiration is impeded and bleeding increases. The largest bored tube which can be passed comfortably should be used.

If the metal endotracheal connection is too narrow it will obstruct respiration

The endotracheal tube may become kinked <sup>(5)</sup> This complication is especially liable to happen when a soft tube is passed orally and such a tube can also be compressed by a throat pack. A tube passed nasally can kink. This dangerous accident is liable to occur when the metal connection is attached to the tube after it has been passed into the trachea. If the metal connection is then rotated so as to lie in the desired position on the face the tube may become twisted within the nose well out of sight. A kink so produced may distort the tube enough to obstruct respiration partially or completely.

An endotracheal tube may become kinked if it is not firmly fixed in position in nose or mouth. The tube may then slip <sup>(6)</sup> out a short distance during anæsthesia, and the heavy connection and corrugated breathing tubes pulling on it may bend it enough to obstruct the airway.

These potentially grave errors are easily rectified provided the anæsthetist is aware of them. Better still they are easily avoided if the tube is well placed and fixed in position before the head is draped in towels.

<sup>(7)</sup> A tube which is too long will enter a main bronchus usually the right which branches off the trachea at a smaller angle than does the left main bronchus. This accident is particularly liable to occur in children, where the distance between the vocal cords and the bifurcation of the trachea is small. Fortunately this state of affairs is easily recognised. One side of the chest moves freely while the other does not. If a stethoscope is used breath sounds are heard on the one side, and not on the other. Respirations become rapid. There is tachycardia usually cyanosis, and lightening of anæsthesia. This accident is easily corrected by gently withdrawing the tube until these signs disappear which they do immediately, provided the condition is recognised and remedied before the lung temporarily out of action has collapsed.

Lastly it must not be forgotten that a tube can become <sup>(8)</sup> blocked by blood or mucus. This must be aspirated at once and where this cannot be done the tube must be changed even if the operation has to be held up temporarily.

## LARYNGOSCOPES

Whatever laryngoscope is used the principles of laryngoscopy remain the same. If these—namely correct head position and adequate anæsthesia—are ignored no laryngoscope will give success. The



secret of successful intubation lies with the anæsthetist, not with any particular laryngoscope. That good results can be obtained with many different patterns is obvious from the fact that these different types all have their supporters.

The standard laryngoscopes vary chiefly in the angle made by the blade with the handle and in the layout of the lighting system. The instrument shown in the foregoing diagrams is that advocated by Guedel who favours an acute angle between handle and blade.

From among the many laryngoscopes available we single out descriptions of two in which we are especially interested. That difference in design is not an all important matter is suggested by the fact that the authors are not agreed as to which is the better.

The Macintosh laryngoscope<sup>8</sup> is designed so that the tip of the blade comes to rest in the angle between the base of the tongue and the

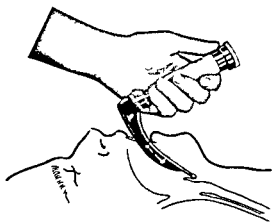


FIG 123

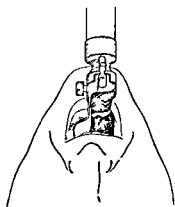


FIG 124

epiglottis (fig 123). The actual shape of the blade is of secondary importance<sup>9</sup> but the curved blade illustrated is recommended. Fig 124 shows the view down the laryngoscope at this stage: the front of the epiglottis is seen. When the handle is lifted in the direction of the arrow (fig 125) the base of the tongue is forced upwards. The epiglottis because of its attachment to the base of the tongue is drawn upwards and the larynx comes into view (fig 126).

It should be noted that the blade does not come into contact with the posterior surface of the epiglottis where the innervation is the superior laryngeal nerve: the stimulation of which results in adduction

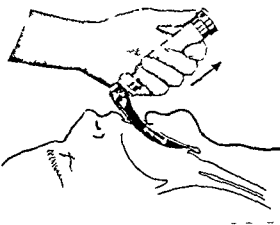


FIG 12a



FIG 12b

of the cords. The end of the blade touches only areas innervated by the glossopharyngeal nerve which plays no part in this reflex. On this account the larynx can be exposed at a lighter plane of anaesthesia than when a standard laryngoscope is used but this is not necessarily a great advantage.

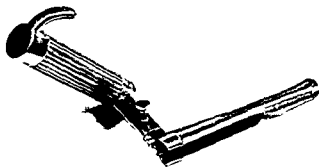


FIG 127

The laryngoscope shown above was devised by Macbeth and Bannister<sup>10</sup>. The handle is set eccentrically so that the left hand can be well out of the way thus allowing more room for manipulations with the right hand.

The blade is easily removed for sterilisation. The curves of the blade are so designed that it picks up the epiglottis easily and without

injury to the pharynx. The illumination is by one of the twin lighting bulbs of the Negus instrument which does not become hot when the lamp is in use. The bulb can be boiled so that blade and bulb form a sterilisable unit.

The obtuse angle between blade and handle was adopted after trial at all angulations from the perfectly straight instrument (blade and handle in a straight line) to the very acute angled and was finally decided upon because it appeared least easy with this angulation to exert leverage on the teeth.

### REFERENCES

- (1) Jackson C and Jackson C L 1934 *Bronchoscopy Esophagoscopy and Gastroscopy* Philadelphia
- (2) Jackson C and Jackson C L 1937 *The Larynx and its Diseases* Philadelphia
- (3) Bannister F B and Macbeth R G 1944 *Lancet* 651
- (4) Gillespie N A 1941 *Endotracheal Anæsthesia* Madison Wisconsin
- (5) Thomas G J 1938 *Anæsth and Analges* 17 301
- (6) Rovenstine E A 1939 Personal communication
- (7) Pask E A 1940 Personal communication
- (8) Macintosh R R 1943 *Lancet* 1 205
- (9) Macintosh R R 1944 *Lancet* 1 485
- (10) Macbeth R G and Bannister F B 1944 *Lancet* 654

## CHAPTER XXVI

MOUTH-GAGS, PROPS, PACKS, AND  
TONGUE-FORCEPS

## MOUTH-GAGS

A GAG is an instrument used for opening the mouth and for keeping it open. There are many different types of gags and in some hospitals all are called by the name of Mason the surgeon who introduced the basic model about 1870.<sup>1</sup>

An anæsthetist's equipment should include two or preferably three good gags. They last a lifetime and the question of economy should not enter into their purchase. The mechanism by which an open gag is locked and released varies with different models and an anæsthetist usually tries many before making his final choice. It is essential that the construction of the gag should be such that it can be opened and closed easily and smoothly by simple movements of one hand.

The skill of a dental anæsthetist can often be judged by the way he handles a gag and allows the dentist easy access to any part of the mouth. Insertion and manipulation of the gag should be unhurried and gentle except in the rare case in which the mouth has to be opened because a patient is *in extremis*.

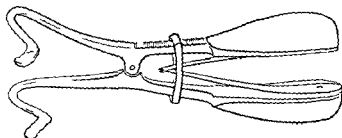


FIG. 128

The ratchet control of this gag frequently referred to as Fergusson's makes it steadier and more controllable than any other. The ratchet-lock is easily adjusted by the thumb and forefinger of the hand which holds the gag and once in position will not slip unless the handles are approximated.

Any gag can be supplied either with ordinary or with Ackland's

injury to the pharynx ~~The illumination is by one of the twin lighting bulbs of the Negus instrument which does not become hot when the lamp is in use~~ The bulb can be boiled so that blade and bulb form a sterilisable unit

~~The obtuse angle between blade and handle was adopted after trial~~ at all angulations from the perfectly straight instrument (blade and handle in a straight line) to the very acute angled and was finally decided upon because it appeared least easy with this angulation to exert leverage on the teeth

### REFERENCES

- (1) Jackson C and Jackson C L 1934 *Bronchoscopy Esophagoscopy and Gastroscopy* Philadelphia
- (2) Jackson C and Jackson C L 1937 *The Larynx and its Diseases* Philadelphia
- (3) Bannister F B and Macbeth R G 1944 *Lancet* 651
- (4) Gillespie N A 1941 *Endotracheal Anæsthesia* Madison Wisconsin
- (5) Thomas G J 1938 *Anesth and Analges* 17 301
- (6) Rovenstine E A 1939 Personal communication
- (7) Pask E A 1940 Personal communication
- (8) Macintosh R R 1943 *Lancet* 1 205
- (9) Macintosh R R 1944 *Lancet* 1 485
- (10) Macbeth R G and Bannister F B 1944 *Lancet* 654

of more ethyl chloride has produced the desired depth of anaesthesia, and then is opened

If the jaws become clenched during  $N_2O$  anaesthesia it is generally impossible to relax them by deepening the anaesthesia since the increasing asphyxia will in fact intensify the spasm. In these cases, it is actually necessary to give oxygen to reduce the asphyxia before an attempt is made to insert the gag.



FIG 132

We disapprove of the use of a boxwood wedge (p 276) for an unconscious patient because it is liable to break teeth when it is forced between the jaws.

The anaesthetist steadies the forehead with one hand and by continuous pressure on the chin depresses the mandible with the other (fig 132). At the same time the dentist retains the nose piece in position and inserts the gag as soon as this can be done.

When the gag has been introduced, the anaesthetist releases his hold on the forehead and chin and steadies the gag. A second gag is then inserted on the other side of the mouth and by exerting steady and



FIG 133

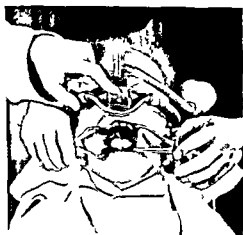


FIG 134

continuous pressure with both gags the masseteric spasm is overcome. If uneven or unnecessary force is applied periostitis may result even

blades The former are curved and lie one on top of the other and although difficult to insert unless the jaw is relaxed they will when

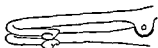


FIG 129

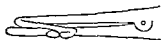


FIG 130

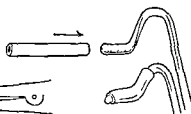


FIG 131

once in position both open and keep the mouth open without slipping

Ackland's blades (fig 130) are straight and since when the gag is closed they lie in the same plane they can be more easily inserted in cases in which the jaw is clenched Once they are in position however there is a tendency for them to slip off the teeth

The blades should be covered with rubber tubing to minimise the risk of chipping the teeth or injuring the soft tissues If the tubing has cracked or become loose it must be replaced by fresh tubing to obviate the danger of its slipping off the blades and becoming a foreign body

The anaesthetist should be on the watch to see that a gag is not handed to him straight out of the steriliser—otherwise he may burn his fingers or still worse the patient's cheek and mouth When the gag is being inserted the lips must be well separated to avoid their being pinched between the gag and the teeth (fig 135) When this precaution is omitted the lip may be bruised or even perforated The gag should be placed as far back in the mouth as possible to avoid bearing on single rooted teeth Bridges, crowns, and loose or fragile teeth must be most carefully avoided, and with children it must be remembered that a deciduous tooth can be dislodged easily

### Use of the Gag to open a Clenched Jaw in an Unconscious Patient

During induction of anaesthesia the prop may slip or be spat out the latter especially in children or it may slip during extractions In children under ethyl chloride the gag may be introduced by the method illustrated below or if the spasm is particularly resistant the mask must be removed until the anaesthesia has become so light that the child opens his mouth to cry as he always does before he recovers consciousness whereupon the anaesthetist inserts the gag The gag with the blades still closed is then held in place until the administration

thetia after the change is made. Hence though anæsthesia has been adequate for extractions on the one side if the anæsthetist then inserts the gag on this side and roughly forcēs the mouth open to allow the prop to be removed anæsthesia may lighten sufficiently to cause mouth breathing even though the mixture of gases administered remains unaltered. Mouth breathing will not, however be initiated if the gag is used gently.

### *Use of a Gag in a more or less Edentulous Patient*

Such a patient usually finds a prop uncomfortable and liable to slip about on the ridge of the gum. Since a gag is easily introduced between jaws lacking in teeth, many anæsthetists often induce anæsthesia in such a patient without having inserted the usual prop opening the mouth with a gag after unconsciousness is attained.

### **Holding a Gag in Position**



FIG 137



FIG 138

These photographs show the right and wrong ways of retaining a gag in position. The gag is self locking so that the anæsthetist by placing the palm of the hand over it and holding the jaw forward can steady the head as easily as if the gag were not there. The shank of the gag is pressed firmly against the cheek to prevent the blade slipping off tooth or gum into the sulcus between the gum and cheek. It is wrong to keep the gag open by holding the handles for then the anæsthetist loses control of the situation the head cannot be steadied and any movement of the head may dislodge the teeth on which the blades rest.



when the teeth are sound also if the blades of the gags rest directly on alveolar mucous membranes these may be badly bruised Any such damage may cause much more after pain than arises from the extraction sockets

Once the mouth has been opened sufficiently it can be kept open easily by one gag only or a prop can be inserted and both gags withdrawn

### Use of a Gag to make either Side of the Mouth Accessible

After extractions on one side have been completed the gag is inserted on this side and opened just sufficiently to release the prop from the opposite side

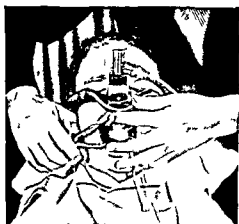


FIG 135



FIG 136

Extractions on the right side have been finished and the left side is to be made accessible

When the prop is released the gag can be used to keep the mouth open or the prop can be reinserted just in front of the gag and the gag taken away Usually it makes little difference which procedure is followed but many dentists prefer to have the prop reinserted because the gag keeps the lips tightly stretched and access to the back teeth of the opposite side is limited

A sudden forceful separation of the jaws by a gag often lightens the anæsthesia sufficiently to cause a reversion from nose to mouth breathing a fact which may be demonstrated when making a change Lack of appreciation of the severity of this stimulus accounts indeed for many of the failures to maintain smooth anæ-

violent particularly in a nervous patient. Hence it is generally advisable to insert a prop between the teeth before commencing to give an anæsthetic for dental extractions whatever anæsthetic is to be used. If this is not done time will be lost, and damage may be done in forcing the mouth open afterwards.

The ideal prop should be neat, comfortable to bite on, and not liable to slip. It should be easy to clean and sterilise and not liable to collect debris. The following diagrams illustrate the props in common use.

**Hewitt's prop**, (fig 140) introduced in 1891 was an improvement on its predecessors inasmuch as it is so shaped as to adjust itself to the angle made between the upper and lower jaws when the mouth is open. Its disadvantages



FIG 140

are that it is tedious to clean and that blood is liable to collect under the rubber cap where it is easily overlooked.

**Trewby's lead-covered prop** (fig 141) is popular in hospital practice, where it is usual to find a set with each prop connected by a chain to a common

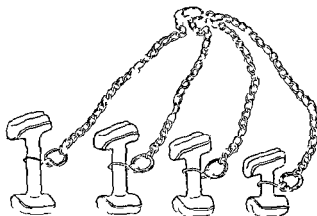


FIG 141

ring. The prop is easily cleaned and sterilised and is everlasting.

**The Trewby prop with rubber insets** (fig 142) on the biting surfaces is more satisfactory but more expensive. Six different sizes are made. The prop is neat and comfortable and is easily cleaned and sterilised. Also the rubber insets which are easily renewable fit tightly so that blood cannot collect under them.

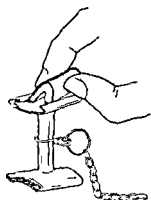
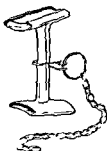


FIG 142.

### Trismus

In a case of trismus where it is necessary to extract the tooth causing this condition a gag should be inserted before anæsthesia is commenced since if the spasm is severe it will be found almost impossible to do this later under light anæsthesia such as commonly is administered in the dental surgery

Separation of the teeth sufficiently to allow the insertion of the gag often is accomplished only after exercising much patience and usually causing some discomfort. The forehead should be steadied, and the chin depressed gently but firmly and continuously for sudden or jerky attempts to separate the jaws cause pain and increase the spasm. At the same time the patient may be given a boxwood wedge mouth opener so that he may help to get his teeth sufficiently far apart for introduction of the gag. With four or five minutes persistence even the most unpromising case yields to this treatment.

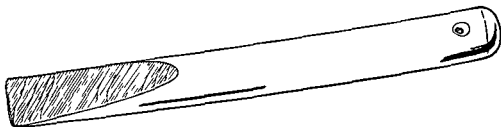


FIG 139

A boxwood mouth opener should be used only by the patient so ensuring that sufficient force will not be used to break his teeth.

Once the gag is in position anæsthesia is induced a second gag is then inserted on the opposite side and the masseteric spasm overcome by steady continuous pressure on both gags.

Unless the teeth can be separated enough for the blades of a gag to be inserted while the patient is conscious an anæsthetic should not be given in the dental surgery. We have never met such a case but should one be encountered we advise postponing the operation until deep anæsthesia can be attained in surroundings where tracheotomy could be performed if the patient vomited.

### MOUTH-PROPS

It is well recognised that there may be spasm of the masseter muscles even after surgical anæsthesia has been established and other muscles are well relaxed. The intensity of the spasm varies and during the light anæsthesia common in the dental surgery it is not infrequently

violent particularly in a nervous patient. Hence it is generally advisable to insert a prop between the teeth before commencing to give an anæsthetic for dental extractions, whatever an esthetic is to be used. If this is not done time will be lost and damage may be done in forcing the mouth open afterwards.

The ideal prop should be neat, comfortable to bite on and not liable to slip. It should be easy to clean and sterilise and not liable to collect debris. The following diagrams illustrate the props in common use.

**Hewitt's prop**, (fig 140) introduced in 1891, was an improvement on its predecessors inasmuch as it is so shaped as to adjust itself to the angle made between the upper and lower jaws when the mouth is open. Its disadvantages

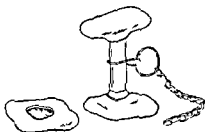


FIG 140

are that it is tedious to clean and that blood is liable to collect under the rubber cap where it is easily overlooked.

**Trewby's lead-covered prop** (fig 141) is popular in hospital practice where it is usual to find a set with each prop connected by a chain to a common

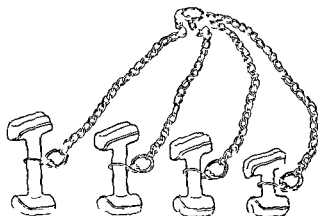


FIG 141

ring. The prop is easily cleaned and sterilised and is everlasting.

**The Trewby prop with rubber insets** (fig 142) on the biting surfaces is more satisfactory but more expensive. Six different sizes are made. The prop is neat and comfortable and is easily cleaned and sterilised. Also other rubber insets which are easily renewable fit tightly so that blood cannot collect under them.

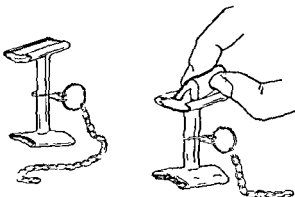


FIG 142.

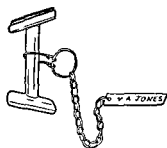


FIG 143

Mushin's prop is similar to Trewby's except that the two biting plates are set at equal angles to the connecting bar. It is claimed that this arrangement makes the prop less liable to slip and is hence particularly advantageous for an edentulous patient.

The de Pass prop, made of solid rubber, is bent double on the thin flexible centre portion before being inserted into the mouth. The ridges on the biting surfaces of the prop are gripped by the teeth and the tramlines formed by the raised

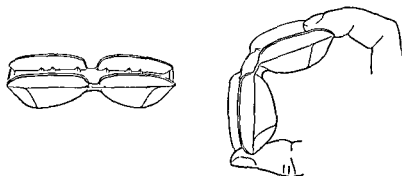


FIG 144

borders of its biting surfaces prevent it from slipping laterally in a toothless patient; however, it is not easily retained.

Its resilience makes it particularly useful for if the patient yawns for example during induction with thiopentone or if during the operation the lower jaw is depressed the prop opens and accommodates itself to the temporarily increased opening of the mouth and does not

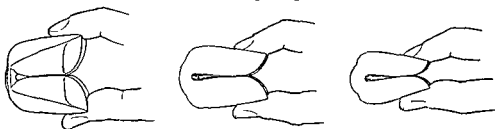


FIG 145

fall out. It is accordingly especially useful in major dental operations where anaesthesia is often deep enough to relax the masseter muscles.

The prop is made in three sizes. Except that for a conscious patient the largest is rather a mouthful, we find this size extremely

satisfactory, but each of the two smaller sizes in our experience is dislodged easily, for when bent into position its two halves are not in contact throughout their whole length, and any biting pressure is liable to twist the prop

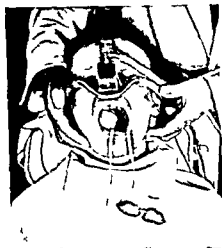


FIG 146



FIG 147

Manipulation of two de Pass props joined by a tape allows both sides of the mouth to be made accessible without the use of a gag. After extractions on the right side are completed the second de Pass



FIG 148



FIG 149

prop is inserted on that side to provide access to the teeth on the left. This singularly easy way of effecting a 'change' has the great advantage that it obviates the possibility of damage by a gag

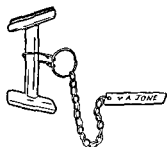


FIG 143

Mushin's prop is similar to Trewby's except that the two biting plates are set at equal angles to the connecting bar. It is claimed that this arrangement makes the prop less liable to slip and is hence particularly advantageous for an edentulous patient.

The de Pass prop, made of solid rubber, is bent double on the thin flexible centre portion before being inserted into the mouth. The ridges on the biting surfaces of the prop are gripped by the teeth and the tramlines formed by the raised

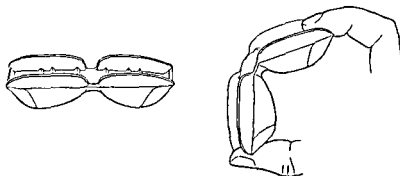


FIG 144

borders of its biting surfaces prevent it from slipping laterally, in a toothless patient however it is not easily retained.

Its resilience makes it particularly useful for if the patient yawns for example during induction with thiopentone or if during the operation the lower jaw is depressed the prop opens and accommodates itself to the temporarily increased opening of the mouth, and does not



FIG 145

fall out. It is accordingly especially useful in major dental operations where anaesthesia is often deep enough to relax the masseter muscles.

The prop is made in three sizes. Except that for a conscious patient the largest is rather a mouthful we find this size extremely

satisfactory but each of the two smaller sizes in our experience is dislodged easily for when bent into position its two halves are not in contact throughout their whole length and any biting pressure is liable to twist the prop



FIG 146



FIG 147

Manipulation of two de Pass props joined by a tape allows both sides of the mouth to be made accessible without the use of a gag. After extractions on the right side are completed the second de Pass



FIG 148



FIG 149

prop is inserted on that side to provide access to the teeth on the left. This singularly easy way of effecting a change has the great advantage that it obviates the possibility of damage by a gag



**Brunton's swivel prop** is inserted between the incisor teeth and it swivels as illustrated. After extractions on one side of the mouth are completed the other side is made accessible very easily by swinging the vertical bar through half a circle. It is especially useful when



FIG 150

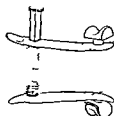


FIG 151



FIG 152

extractions on both sides have to be undertaken for children for with children one may anticipate anæsthetic difficulties which will reduce the operating time available. Some dentists condemn this prop for fear that it will damage the incisor teeth but we have not seen or heard of any injury so caused.

### Further Points concerning Props in General

The chances of a prop slipping when the patient is largely edentulous are lessened by previously directing him to free his mouth from mucus by thorough rinsing. If the prop does slip it is usually easy with such a patient to open the mouth with a gag.

The mouth should never be propped open more widely than necessary since this makes the patient uncomfortable and makes it difficult for him to breathe through his nose (p. 296). For a nervous patient a very small prop is sometimes inserted and the mouth opened more widely after he has become unconscious. With a little care it is possible to select a prop which will allow adequate access and at the same time be comfortable.

A child may attempt to spit the prop out or it may fall out if he cries or struggles. To keep it in position upward pressure must be applied to the chin immediately it is inserted.

A nervous patient may refuse a prop or may begin to retch as soon as it is inserted in which case the prop should be removed at once or he will be restive throughout the whole of induction. Nitrous oxide is given nasally and the patient is told that a few breaths of gas will make his throat less sensitive. After about six breaths of  $N_2O$  the

patient will be sufficiently amenable to suggestion will open his mouth when requested and will have no difficulty in retaining the prop

## MOUTH-PACKS

### Sponges

During any dental extraction no matter how trivial the oral cavity must be packed off as efficiently as possible to prevent any foreign body being inhaled or swallowed. The ideal packing material is still a matter for discussion but we ourselves find that a bleached coarse meshed sponge about as large as a small orange is the most satisfactory. Bleaching improves the appearance, and the extra light reflected from the bleached sponge helps to illuminate the field of operation. The coarseness of the mesh is essential for without it the sponge would be less capable of absorbing blood and saliva, or of entangling any stray fragments of teeth.

A sponge is difficult to sterilise and it is most satisfactory to use each only once. If however for economy the same sponges are to be used repeatedly the most efficient method of sterilising them is to wash them in sodium bicarbonate solution and then soak them in a weak solution of lysol for forty eight hours and finally wash them thoroughly with water. Sponges should never be boiled otherwise they will shrink and become inelastic.

The necessity for a mouth pack is discussed on p 358. A sponge becomes an elastic and efficient pack only when moist and it should therefore be squeezed out in cold water before being introduced. If during a prolonged operation the sponge becomes coated with blood and saliva it is a potential danger unless it has a tape attached (p 297). When sodden it should be removed and replaced by a fresh sponge after the mouth has been mopped dry.



FIG 153

The sponge with tape attached ready to be introduced into the mouth

**Brunton's swivel prop** is inserted between the incisor teeth and it swivels as illustrated. After extractions on one side of the mouth are completed the other side is made accessible very easily by swinging the vertical bar through half a circle. It is especially useful when



FIG 150

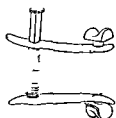


FIG 151



FIG 152

extractions on both sides have to be undertaken for children, for with children one may anticipate anæsthetic difficulties which will reduce the operating time available. Some dentists condemn this prop for fear that it will damage the incisor teeth but we have not seen or heard of any injury so caused.

### Further Points concerning Props in General

The chances of a prop slipping when the patient is largely edentulous are lessened by previously directing him to free his mouth from mucus by thorough rinsing. If the prop does slip it is usually easy with such a patient to open the mouth with a gag.

The mouth should never be propped open more widely than necessary since this makes the patient uncomfortable and makes it difficult for him to breathe through his nose (p. 296). For a nervous patient a very small prop is sometimes inserted and the mouth opened more widely after he has become unconscious. With a little care it is possible to select a prop which will allow adequate access and at the same time be comfortable.

A child may attempt to spit the prop out or it may fall out if he cries or struggles. To keep it in position upward pressure must be applied to the chin immediately it is inserted.

A nervous patient may refuse a prop or may begin to retch as soon as it is inserted in which case the prop should be removed at once or he will be restive throughout the whole of induction. Nitrous oxide is given nasally and the patient is told that a few breaths of gas will make his throat less sensitive. After about six breaths of  $N_2O$  the



FIG 155

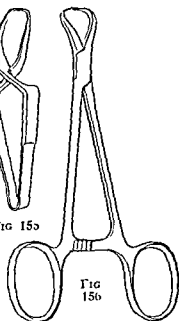
FIG  
156

FIG 157



vessels on the ventral aspect of the tongue they should not be inserted too deeply

These forceps (figs 158 159) are illustrated only to condemn them

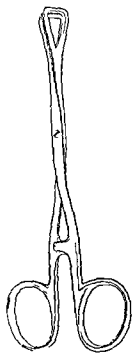


FIG 158



FIG 159

After anæsthesia has been established the sponge is gently pushed towards the back of the mouth until it just touches the front of the soft palate. Incidentally we may mention that in this position where it partly occludes the mouth but does not occlude the pharynx the sponge ensures nasal breathing in the unusual patient who has a short soft palate and uvula and who would otherwise continue to breathe partially through the mouth even during surgical anæsthesia. If the sponge is pushed too far back it may cause retching and restlessness and even obstruct respiration (fig 183 p 297). Though the sponge may seem to fill the mouth it is not a nuisance to the dentist since he can easily push to one side any part of it encroaching on the tooth to be extracted.

In a case of advanced pyorrhœa special care must be taken not to dislodge a tooth while inserting the sponge. Cases are known where teeth have thereby been pushed down into the trachea.

With an anæsthetic-resistant patient the sponge is sometimes soaked in ether before it is inserted (p 117). If with such a patient ethyl chloride is sprayed over the mouth to supplement  $N_2O$  the sponge may become frozen and lose its absorptive properties and a fresh sponge must then be substituted before extractions are begun.

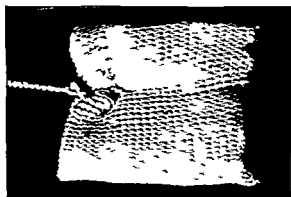


FIG 154

### Cotton - wool Mouth packs

A cotton wool mouth pack is convenient but lacks the elasticity of a sponge. Although effective in absorbing moisture its volume is much more

reduced by compression and it then affords but little protection.

### TONGUE-FORCEPS

Tongue-forceps (fig 155 and fig 156) are sharply pointed so that while they hold the tongue firmly they cause minimal damage. They should be inserted into the dorsum of the tongue at about the junction of the anterior one third and posterior two thirds as illustrated in fig 157 and in fig 175 p 292. The bite of the forceps should be sufficient to prevent them tearing out of the tongue. To avoid damaging the

## CHAPTER XXVII

## CARE OF THE UNCONSCIOUS PATIENT

FROM the moment an anæsthetic is given until the patient becomes rational again his safety is in the hands of his attendants, since his protective reflexes are abolished or depressed. For this reason a basal anæsthetic should not be given unless the patient can be supervised in the pre- as well as in the post operative period. Ideally, during recovery the patient should not be left unattended until he has regained consciousness. During the time the laryngeal reflexes and the respiratory centre remain depressed the airway must be maintained clear and the patient kept in a position in which any foreign body in the pharynx will drain out of the mouth and not be inspired.

Before anæsthesia is induced dentures should be taken out and the mouth examined for loose or fragile teeth. Jewellery should be removed to prevent it being damaged and from cutting into the flesh. If for sentimental reasons the patient does not wish rings to be removed they can be left until unconsciousness has developed.

## Position on the Operating Table

Before the operation is begun the patient's arms must be placed in a safe position conveniently out of the surgeon's way. If the arm hangs over the edge of the operating table the musculospiral nerve may be subjected to pressure and cause temporary paralysis of the muscles it supplies.



FIG 160



FIG 161

A suitable position for the arm is achieved by slightly raising the buttock, placing the fully extended arm palm downwards on the table and securing it by allowing the buttock to roll back on the hand

absolutely on the ground that they crush the tongue and interfere with its circulation. If in emergency tongue-forceps are not available the tongue may be wiped dry and withdrawn by the fingers covered with gauze or a cloth or it can be transfixed with a safety pin and drawn forward.

#### REFERENCES

- (1) Mason F 1876 *Brit med J* **1** 117
- (2) Hewitt F W 1891 *Brit J dent Sci* **34** 37

bed. When the patient is to be transferred, the lifters 'preferably three should all stand on the same side of the trolley in the angle formed by it with the bed. The main weight will be borne by the middle one of the three lifters and by the one at the head while the one supporting the legs needs merely to control them. The lifters' arms are placed well under the patient.



FIG 166

As soon as he has been raised off the bed the patient should be rolled on to his side facing the lifters. In this position the leverage on the arms is small and the patient can be carried in comfort

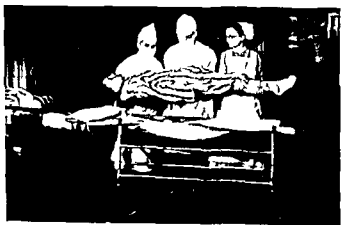


FIG 167

The *wrong* method of carrying a patient. As the patient has not been rolled on to his side he must be supported at arms' length. This is an extremely fatiguing task and if the patient is heavy it may be an impossible one.





FIG 162



FIG 163



FIG 164

An alternative method of securing the arms is to place a towel or folded sheet under the patient, then over and round the arms tucking it in beneath the mattress

Except in so far as the position for operation necessitates care must be taken that no joints are kept in an abnormal position or the ligaments will be stretched and the patient will complain of pain for some time after the operation

When administration of the anæsthetic is stopped the anæsthetist retains responsibility for the unconscious patient until he has passed this on to some other competent person The transfer of responsibility should be quite definite not just a casual understanding, for instance the anæsthetist handing the patient over to a nurse should say,

Nurse can you be responsible for the patient now? and should not be satisfied until she answers with a definite Yes

Before beginning the journey back to the ward with an unconscious patient the person in charge should be satisfied that the airway is perfectly clear and that the patient is properly protected against cold A gag tongue-forceps airway swabs and swab holder a towel and

a bowl should be to hand on the trolley, and should be kept by the bedside until the patient has recovered

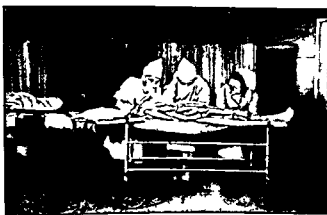


FIG 165

### Lifting the Unconscious Patient

The head of the trolley should be directed towards the foot of the bed or the foot of the trolley to the head of the

unconscious patient should *not* be placed particularly if he cannot be kept under constant supervision. The tongue may fall back or vomitus be aspirated so that death from respiratory obstruction may result within a very short period (fig 219 p 332)

### Protection from Hot Bottles

A hot water bottle in contact with an unconscious patient may produce a serious burn. Hot water bottles should be supplied with flannel covers and before being put near the patient they should as an extra precaution be wrapped in a blanket.

### Vomiting

The danger of vomiting during the unconscious period is that of inhalation of vomitus causing immediate respiratory obstruction or more remotely, pulmonary complications.

During both induction of and recovery from anæsthesia the patient passes through a stage at which vomiting may occur. The incidence

of vomiting during induction depends considerably on the skill of the anæsthetist and can be eliminated by making induction smooth and rapid thus keeping the patient for as short a time as possible at the vomiting level. Normally the patient properly prepared for operation has an empty stomach by the time anæsthesia is begun nevertheless regurgitation of acid gastric juice may occur and its local irritant action on the larynx cause troublesome laryngospasm. The patient on whom an emergency operation is being performed may



FIG 171

### The Patient's Position in Bed

After being lowered on to his bed the patient is placed in the semi prone position facing the light. To prevent him from rolling



FIG 168

backwards his lower arm is drawn behind him. A firm pillow tucked into the angle between his chest and the bed prevents him from rolling on to his face. Flexion of the upper leg assists in fixing the patient in a

position in which the tongue falls away from the pharynx so that the airway remains clear and vomitus, blood or mucus drain freely out of the mouth (fig 215 p 330)

The presence of blood in the mouth makes this position particularly desirable after operations on the mouth or throat



FIG 169

Once the patient has recovered sufficiently he can be allowed to assume any position in which he feels comfortable though he should not be left unattended until he is responsible for his actions



FIG 170

This photograph is included to show the position in which the

## CHAPTER XXVIII

### RESPIRATORY OBSTRUCTION

INTERFERENCE with the passage of gases between the anæsthetic mask and the lungs is the commonest cause of anæsthetic difficulties and disasters. The penalties of obstruction vary with its degree of completeness and with the patient's condition. They range from mere disturbance of the smoothness of the anæsthesia to the death of the patient.

The most common site of obstruction is the small space between the base of the tongue and the rima glottidis. The more usual causes can be classified into four groups.

- 1 Backward displacement of the tongue
- 2 A foreign body, e.g. sponge, vomitus, tooth
- 3 Adduction of the vocal cords caused by anoxial spasm of the laryngeal muscles
- 4 Œdema of the aryepiglottic folds, due to spread of infection from neighbouring structures

Obstruction in the first three groups occurs as an accident associated with unconsciousness. The fourth group exists pre-operatively and as will be seen later, this condition is a contra-indication to the administration of a general anæsthetic.

#### BACKWARD DISPLACEMENT OF THE TONGUE

Backward displacement of the tongue is the commonest cause of respiratory obstruction in an unconscious patient. The problem is one of simple mechanics. It occurs only when the tongue falls or is pushed backwards. The former is likely to happen when the patient is lying on his back; the latter during operations on the lower jaw, no matter what the position of the patient may be. When the patient is lying on his back it is particularly easy for the tongue and the lower jaw to which it is attached to fall backwards. The tongue, as Chevalier Jackson<sup>1</sup> points out, acts as a 'check valve' at the laryngeal opening, obstruction to expiration being negligible. The tongue is forced forwards by each expiration, after which it immediately falls back into its gravity position again and attempts at inspiration draw it over the laryngeal entrance and obstruct ingress

have a stomach full of food if he vomits acute respiratory obstruction can occur easily unless he is immediately placed in a position which allows the vomitus to drain away. The head should be lowered by removing any pillows by lowering the head rest (fig 171), or by tilting the operating table into the Trendelenburg (head down) position. Drainage is further assisted by turning the head to one side and better still by rolling the patient as far as practicable on to one side. If necessary the mouth must be opened by a gag and debris cleared away by swabs held in holders.

The method of dealing with vomiting during anæsthesia in the dental chair is described on p. 233.

### Post-operative Restlessness

Restlessness during recovery from anæsthesia may be due to <sup>(1)</sup> oxygen want a condition which is usually readily recognisable and is especially apt to occur in an exsanguinated patient. Much more frequently it is a response to pain from the site of operation at a time when the patient has not yet recovered consciousness. This is frequently seen after the use of some basal anæsthetics the properties of which are more hypnotic than analgesic. When these have been used the patient remains for a long time in the stage of potential delirium and whether he is restless or not depends on whether he is exposed to pain or not. The degree of restlessness varies with the patient's nervous make up and with the severity of the pain and ranges from the mere occasional changing of his position in bed to maniacal excitement. The treatment is to give morphia since this drug acts selectively on perception of pain. It should be noticed that post operative restlessness does not occur unless the morphia given as a premedicant has been excreted before painful stimuli from the operation have ceased.

A simple rule for the administration of morphia to the unconscious patient recovering from an operation is that it should be given if he is restless but not otherwise. Since restlessness is almost invariably a response to painful stimuli it is a sign that the patient has recovered sufficiently to make the administration of morphia safe. In the early post operative period even  $\frac{1}{2}$  gr. has a marked sedative effect and is usually sufficient to quieten an adult. The length of time for which a given dose of morphia will be effective depends on the metabolic rate of the patient and on the intensity of the pain. The influence of a raised metabolic rate is seen during the recovery from any operation of a patient suffering from exophthalmic goitre a second injection of morphia may be needed half an hour after a dose which would have been adequate for several hours had the metabolic rate been normal.

Attempts at artificial respiration when the tongue is occluding the larynx are worse than no attempt at all, since the check valve (p 291) allows air to pass in one direction only—from within outwards.

When the patient is sitting upright in the position usual for dental extraction the tongue is not an obstruction to respiration—it becomes one only if it is pushed backwards by the dentist. During the extraction of lower back teeth it is particularly easy for the dentist to make the mistake of steadying the mandible by pressing it backwards and downwards in such a manner as to obstruct respiration by forcing the base of the tongue against the posterior pharyngeal wall.



FIG 176

*Radiograph*—The dorsum of the tongue has been painted with barium paste. The epiglottis is readily distinguishable.

Freedom of the airway is ensured by the dentist pulling the mandible forwards and upwards with his left hand. The anaesthetist helps by forward pressure on the angle of the jaw. Compare the plane of the incisor teeth with that seen in fig 177.

of air The patient will die quickly if this condition is not relieved and this relief usually can be attained simply by holding the angle of the jaw forward (fig 178 p 294), thus carrying the tongue away from the laryngeal opening. If this is not sufficient to clear the airway a mechanical airway can be inserted or the tongue pulled forwards by tongue forceps

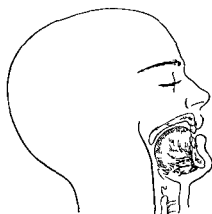


FIG 172 —Tongue in normal position

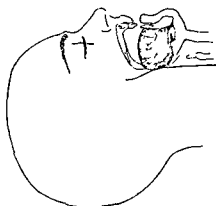


FIG 173 —The airway is completely obstructed by the tongue which has fallen back by gravity

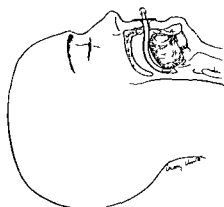


FIG 174 —An airway restorer (usually called an airway) keeps the tongue forward and an uninterrupted path for respiration is restored

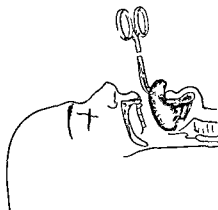


FIG 175 —In an emergency if an airway is not available the tongue can be pulled forward by sharp pointed forceps. If such tongue forceps have by oversight been omitted from the anaesthetist's equipment a towel clip or a large safety pin may be used or the tongue can be wiped dry and covered by a cloth pulled forward by the fingers

Respiratory obstruction may occur also from faulty posture in the dental chair. Either flexion or extension of the head can cause the tongue to occlude the airway.

If the patient sits with the head flexed or if he slips into this position, free respiration is impeded. To obviate this the head rest should be adjusted to the nape of the neck, and the unconscious patient can be prevented from slipping by the support of a strap round the pelvis.



FIG 179



FIG 180

This radiograph shows obstruction of the airway by the tongue when the head is flexed as in fig 179. The obstruction is aggravated if the mandible is pressed backwards or downwards.





FIG 177

Pressure backwards and downwards on the mandible results in varying degrees of respiratory obstruction. This radiograph (fig 177) shows how the mandible can be pushed backwards by the dentist's left hand. The tongue is carried backwards and forced against the posterior pharyngeal wall causing complete obstruction.

Respiration may be impeded simply by the tongue being

made to bulge backwards. Obstruction so caused can occur to some extent even if the lower jaw is held forwards but will obviously be more complete if it is pushed backwards.

The anaesthetist can help the maintenance of a free airway by pressing forward the angle of the patient's jaw. If at the same time he steadies the mandible the dentist is given a firm purchase for his extraction and the tendency for the jaw to be pushed backwards is greatly diminished. Prolonged pressure on the angle of the jaw by the anaesthetist's fingers may cause post operative pain at the site of pressure. The ring finger can feel the pulsation of the carotid artery.



FIG 178

Respiratory obstruction may occur also from faulty posture in the dental chair. Either flexion or extension of the head can cause the tongue to occlude the airway.

If the patient sits with the head flexed or if he slips into this position, free respiration is impeded. To obviate this the head rest should be adjusted to the nape of the neck and the unconscious patient can be prevented from slipping by the support of a strap round the pelvis.



FIG 179



FIG 180

This radiograph shows obstruction of the airway by the tongue when the head is flexed as in fig 179. The obstruction is aggravated if the mandible is pressed backwards or downwards.

If the head rests and the head becomes extended the tongue can act as a check valve as it does with a patient in the supine position (fig 173 p 292)



FIG 181

mouth to its utmost limits carries the base of the tongue on to the posterior pharyngeal wall

If the tongue is relaxed the wider the mouth is opened the more difficult does nasal respiration become. Hence a widely opened mouth favours oral breathing.

An incorrectly adjusted nose-piece may partially occlude the nares and so impede free respiration (fig 76 p 221)

This position has other disadvantages. Blood and mucus gravitate backwards and irritate the posterior pharyngeal wall causing restlessness and retching in the lightly anaesthetised patient. Also the patient is particularly exposed to the danger of entry of a foreign body into the lungs (pp 308 and 362).

A not infrequent cause of minor respiratory obstruction in dental anaesthesia is the insertion of too large a prop. The mouth can be opened only by depressing the mandible and the illustration below shows how opening of the



FIG 182

## FOREIGN BODIES

(A) The sponge or mouth pack, which should invariably be used to protect the larynx when extractions are being performed may cause respiratory obstruction

If the sponge is pushed too far back the soft palate is forced against the posterior pharyngeal wall and blocks the airway partially or completely (fig 183). Nose breathing is prevented by the palatal obstruction and oral breathing by the sponge itself.

A sponge sodden with blood and saliva if compressed suffers a marked decrease in volume and if the small sodden sponge slips behind the base

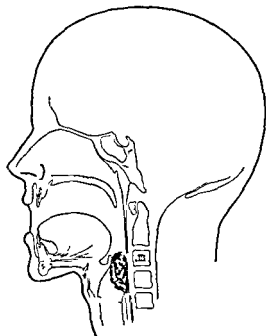
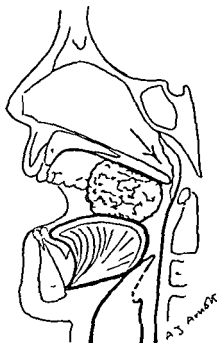


FIG 184

FIG 183 (After Archer<sup>2</sup>)

of the tongue it will completely occlude the laryngeal opening (fig 184). This is a rare accident, but when it happens respiratory arrest is dramatic in its completeness and suddenness. A case of respiratory obstruction from this cause is described on p 314.

(B) Vomitus if inhaled may occlude the larynx.

(C) During every extraction both dentist and anaesthetist must be constantly alert to the possibility of a tooth tooth fragment or other foreign body slipping past the sponge into the laryngeal opening. Despite the undoubted value of a sponge, constant vigilance on

the part of the dentist is essential for protection against this accident

Although the foreign bodies described under (B) and (C) may cause acute respiratory obstruction by lodging at the laryngeal entrance it is more common on account of their small size for them to pass through the rima glottidis and cause grave post operative pulmonary complications

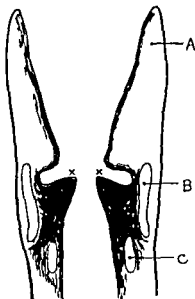
### LARYNGEAL SPASM

Anoxæmic spasm of the laryngeal muscles leading to spastic adduction of the vocal cords is most likely to happen in the unpremedicated thick set muscular individual who often is described as unsuitable for nitrous oxide anæsthesia and who in fact is resistant to every form of anæsthesia. In the frail or heavily premedicated patient muscular spasm is not a feature of oxygen-want. Nitrous oxide is the weakest of all the anæsthetics in common use and if for any reason it is chosen for a resistant patient the range of anæsthesia is necessarily narrow. If the patient is unpremedicated and supplementary anæsthesia is not used its feeble action must be reinforced by a marked reduction of the oxygen intake to prevent the patient responding to stimuli by struggling and mouth breathing. At this low level of oxygen tension anoxæmic spasm of some of the muscle groups is inevitable. The muscles which adduct and abduct the cords like all other muscles of the body work smoothly only when the oxygen supply is adequate. Unco-ordinated action of the laryngeal muscles resulting from anoxæmia is evidenced by stertorous and jerky breathing. With deepening anoxæmia spasm of the laryngeal muscles increases in intensity and since the muscles of adduction are more powerful than those of abduction, the cords eventually become tightly approximated. Throughout the development of this condition expiration is impaired but slightly, but inspiration becomes less and less effective until, with what appears to be dramatic suddenness respiration ceases with the chest in the position of expiration. The mechanisms concerned in the deflation of the thorax will be appreciated by an examination of the following diagrams

The cords during deep ether anesthesia on the left, as seen through a laryngoscope on the right the corresponding coronal section of the



FIG 185

FIG 186 (After Jackson<sup>3</sup>)

A = Aryepiglottic fold

B = Thyroid cartilage

C = Cricoid cartilage

x = Vocal cords

larynx. The cords are in the mean position of normal quiet respiration. Although they become widely abducted in forced respiration the range of movement in normal respiration is slight.



FIG 187

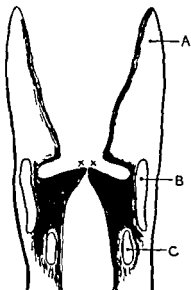


FIG 188

Adduction of the vocal cords in laryngeal spasm

These figures show a more advanced degree of laryngeal spasm during light ether anæsthesia. The arytenoid cartilages are in close



FIG 189

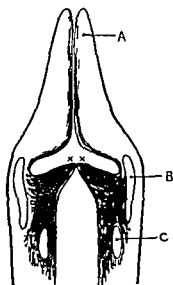


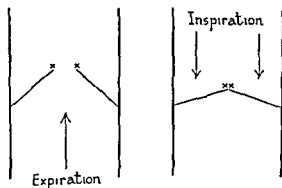
FIG 190

- A = Aryepiglottic fold  
B = Thyroid cartilage  
C = Cricoid cartilage  
XX = Vocal cords

apposition. The adducted vocal cords are covered by the tightly approximated folds of mucous membrane known as the false cords. These in turn are obscured by the aryepiglottic folds, which close the larynx with a sphincter like action. The vertical slit lies over the laryngeal entrance. It will be noticed that the spasm has prevented the blade of the laryngoscope from being carried as far forwards as in fig 187 and hence more of the pharynx is seen. When the muscles of the larynx are in spasm from anoxæmia the appearance is similar except that the pink colour associated with ether anæsthesia is replaced by a slate blue.

The ineffectiveness of attempts at artificial respiration in this stage can be understood from an examination of the adjoining two figures which illustrate diagrammatically the position of the cords during laryngeal spasm.

The bases of the cords are distal to their free ends. The action of the adducted

FIG 191 (After Jackson <sup>4</sup>)

cords is similar to that of a pair of swinging doors which swing one way only. Air can be expelled from the chest because they swing open to allow its egress. Ingress of air is, however, prevented because the cords swing back like two one-way doors. The more powerful the inspiratory effort the firmer the apposition of the cords. The condition is comparable to check-valve obstruction by the tongue. When respiratory effort ceases as a result of this valve-like action, the chest will be in the position of expiration. At this stage attempts to inflate the lungs either by oxygen under pressure (positive pressure proximal to the cords) or by attempts at artificial respiration (negative pressure distal to the cords) must obviously be ineffective.

Illustrative cases are described and the treatment of this condition dealt with on pp 310-313

### OEDEMA OF THE GLOTTIS

The causes of obstruction so far discussed occur during the course of anaesthesia. Respiratory obstruction of varying severity may occur too in the conscious patient. The most common cause for this is spread of infection from a wisdom tooth to the upper margins of the larynx. The aryepiglottic folds become oedematous and encroach on the airway.



FIG 192—Normal larynx as seen in a laryngoscopic mirror



FIG 193 (After Jackson<sup>5</sup>)—Oedema of right aryepiglottic fold showing encroachment on the laryngeal opening. There is slight oedema of the epiglottis and left aryepiglottic fold also

A conscious patient overcomes the early stages of respiratory obstruction by using his accessory muscles of inspiration. Normally the thorax expands with but little muscular effort since air can be drawn in freely. If the glottic aperture is narrowed by inflammation or by a foreign body the resistance to the passage of air is increased.



The extra power provided by the accessory muscles increases the negative pressure within the thorax and is capable of compensating for varying degrees of respiratory obstruction. At first progress of the obstruction may be insidious and the effects of diminished oxygen intake not conspicuous. Relief of the condition, however, is urgent as the time will come generally without warning when the alveolar oxygen tension is reduced below the critical level necessary to keep

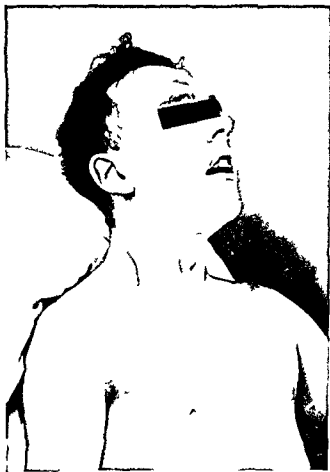


FIG. 194

the patient alert and breathing vigorously. A vicious circle is now established and the patient, who previously was not recognised as being in immediate danger, will die within a few minutes.

This photograph illustrates the clinical picture typical of partial occlusion of the larynx from any cause. Note the anxious expression, the action of the accessory muscles of respiration and the indrawing of the supraclavicular and intercostal spaces. Since air cannot be sucked in freely past the obstruction the negative

pressure within the thorax results in indrawing of the soft parts of the thoracic wall.

When an operation has to be performed for the relief of this condition (e.g. for removal of an impacted wisdom tooth infection round which is causing the spreading œdema) the anæsthetist must realise that anæsthesia will be especially dangerous for a patient who

even when at rest, is obliged to use his accessory muscles of inspiration, for production of unconsciousness in such a patient may cause death. If the obstruction is severe such a patient cannot sleep because as soon as he dozes off the accessory muscles being under *voluntary* control cease to act and he wakes with a sense of suffocation. Similarly if unconsciousness is produced by any general anæsthetic the help of the accessory muscles is lost and if the condition is not dealt with at once the patient will die rapidly of asphyxia. Artificial respiration applied to a patient with such a degree of respiratory obstruction is ineffective and death is inevitable without immediate tracheotomy and artificial respiration.

Because of the patient's respiratory distress the anæsthetist may be tempted to employ a basal anæsthetic the use of which, however, is contra indicated absolutely. The warning against the use of general anæsthetics given in discussing cases of œdema of the larynx applies with particular emphasis to the basal anæsthetics. Respiratory arrest here occurs with the onset of unconsciousness (p. 31) so that the patient is in an even graver condition than would be the normal patient to whom had been given an overdose of basal anæsthetic sufficient to produce respiratory arrest since he has the added grave complication of a quite inadequate airway. Apart altogether from production of unconsciousness no drug should be given which will depress the respiratory centre. It may even be dangerous to give small doses of morphia to relieve the patient's mental and physical distress.

If the degree of obstruction does not necessitate the full action of the accessory respiratory muscles the anæsthetist may feel that the usual respiratory muscles will be capable of achieving just sufficient respiratory exchange provided adequate oxygen is given and therefore that he is justified in administering an anæsthetic without a preliminary tracheotomy. He has only his clinical judgment to guide him here and even if he feels satisfied that the patient can be anæsthetised safely a tracheotomy outfit must be at hand in case of need. If the obstruction necessitates the patient using his accessory muscles to their utmost a tracheotomy must be performed under local anæsthesia before a general anæsthetic is given. Any respiratory difficulty is thus overcome and with it any contra indication to general anæsthesia.

Many years ago one of us was present as an onlooker when an operation was to be performed for the extraction of a wisdom tooth infection round which was causing œdema of the glottis sufficient to make the patient use his accessory inspiratory muscles. Induction of anæsthesia was peaceful but immediately unconsciousness supervened inspiratory effort faded away and despite

The extra power provided by the accessory muscles increases the negative pressure within the thorax and is capable of compensating for varying degrees of respiratory obstruction. At first progress of the obstruction may be insidious and the effects of diminished oxygen intake not conspicuous. Relief of the condition however, is urgent as the time will come generally without warning when the alveolar oxygen tension is reduced below the critical level necessary to keep

the patient alert and breathing vigorously. A vicious circle is now established and the patient who previously was not recognised as being in immediate danger, will die within a few minutes.

This photograph illustrates the clinical picture typical of partial occlusion of the larynx from any cause. Note the anxious expression, the action of the accessory muscles of respiration and the indrawing of the supraclavicular and intercostal spaces. Since air cannot be sucked in freely past the obstruction the negative

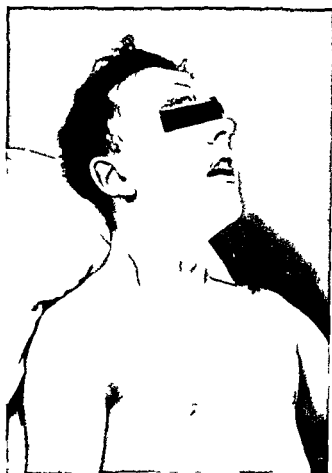


FIG. 194

pressure within the thorax results in indrawing of the soft parts of the thoracic wall.

When an operation has to be performed for the relief of this condition (e.g. for removal of an impacted wisdom tooth infection round which is causing the spreading œdema) the anaesthetist must realise that anaesthesia will be especially dangerous for a patient who

## CHAPTER XXIX

### EMERGENCIES

IN operations in the mouth there are special risks of obstruction to the airway (see previous chapter), and special precautions must be taken. The prevention of anæsthetic emergencies in general will be illustrated here by considering their avoidance in dental anæsthesia in particular. We shall then proceed to consider the different emergencies and their treatment.

All accidents associated with dental anæsthesia should be avoidable, and observance of the following measures will minimise the risk of their occurrence.

1 Before anæsthesia is begun the patient should be settled comfortably in the correct position in the dental chair (fig 61, p 207), and this position maintained by means of a strap round the pelvis.

2 No extensive or prolonged operation should be attempted in the dental chair. Long anæsthesia and extensive dental operations increase the risk of post operative collapse especially in the ambulatory patient and in any case it is difficult without endotracheal anæsthesia to maintain a continuously clear airway.

The preaching of this counsel of perfection is easier than its practice. Hospital beds are rarely available for any except really major dental cases with the result that numerous extensive operations are performed in the out patients department under general anæsthesia. In private practice it is in such cases often difficult to make the patient realise that there are real advantages to be gained from entering a nursing home and that the recommendation to do so despite the expense is not merely a fad of the dentist's. This difficulty is common, and many young practitioners have not the courage to insist on nursing-home treatment. Further unfamiliarity with the strict technique practised in the operating theatre is responsible for the preference of some dental surgeons to operate in their own rooms.

3 Only in exceptional circumstances should anæsthesia in the dental chair be prolonged if the difficulties of the operation are proving greater than were anticipated. It is safer to discontinue the operation and arrange for its completion under local anæsthesia at another session or if necessary in a nursing home or hospital. Prolongation of nasal nitrous oxide anæsthesia merely in order to avoid confession of failure

attempts at artificial respiration the patient died. The underlying pathology was not appreciated and since the patient was thought to be dead from primary heart failure, no tracheotomy was performed although if this had been done promptly it is probable that the patient's life could have been saved.

Deaths from this cause are frequently attributed to the particular anæsthetic used. In turn every anæsthetic has thus been blamed. Not until it is realised that death results from the production of unconsciousness and not from the effect of any particular anæsthetic will the mortality in this type of case be reduced.

#### REFERENCES

- (1) Jackson C and Jackson C L 1937 *The Larynx and its Diseases* Philadelphia and London 181
- (2) Archer W H 1934 *Dent Cosmos* 76, 1233
- (3) Jackson C and Jackson C L 1937 *The Larynx and its Diseases* Philadelphia and London 31
- (4) *Ibid* 270
- (5) *Ibid* 86

Conditions which increase the danger of entrance of foreign bodies into the lungs such as continuing the operation after the operation field has become obscured by bleeding or when the patient is restless, are considered on page 359, where also the importance of packing the throat and the advantages offered by endotracheal anaesthesia for major dental operations are pointed out. Everything in the mouth may be slippery from saliva and, unless the pharynx is well protected, it is all too easy for a foreign body to enter the oesophagus or trachea. It is significant that foreign bodies are lost most often when anaesthesia is turbulent. Fortunately the patient is then so lightly anaesthetised that the swallowing reflex is present, and a foreign body is therefore more likely to be swallowed than inspired. If a foreign body is lost during deep anaesthesia, the risk of its entrance into the larynx is increased because the swallowing reflex has been lost.

In order of frequency the lost foreign body is likely either (a) to pass into the oesophagus, (b) to pass through the glottis or (c) to be arrested by the vocal cords causing acute respiratory obstruction. The correct procedure when a foreign body is missing is dealt with on page 360.

If there is no definite indication whether the foreign body has been swallowed or inspired and if radiological examination is negative the patient should follow at first the simple routine appropriate for a swallowed foreign body but a particularly careful watch must be kept on him for symptoms indicating that it is in the lungs.

**Foreign bodies in the alimentary tract**—It is rare for a swallowed foreign body if it passes through the oesophagus to lodge anywhere in the alimentary canal sufficiently long to do any harm. A jagged foreign body such as a small denture held up in the oesophagus must be removed skilfully as soon as possible by oesophagoscopy under local or general anaesthesia for if there is ulceration of the oesophagus, the infection spreads to the mediastinum and this condition has a very high mortality rate.

If the foreign body is in the stomach or intestines bulky residue foods (e.g. porridge bran) should be given and if thought necessary the faeces may be sifted to discover when it is passed. The temptation to give purgatives to hasten the exit of the foreign body must be sternly resisted since they increase the tendency to spastic contraction and the liability of the foreign body to become impacted an occurrence attended by the risk, otherwise remote of perforation of the intestine. Although the prognosis for a foreign body which has been swallowed is good and surgical interference is rarely necessary that for a foreign

to complete the operation is as risky as performing an extensive operation in the dental chair

4 Nitrous oxide anæsthesia should not be prolonged in patients with physical disabilities which are aggravated by anoxæmia. Patients with toxic myocardial weakness of the post influenzal type and those suffering from exhaustion from overwork, poor feeding or apprehension ideally should be dealt with in hospital or a nursing home and given basal anæsthesia followed by nitrous oxide and ether. Unfortunately for economic reasons they are often subjected to the risks of prolonged anæsthesia in the dental chair

5 Every possible precaution must be taken to prevent the entrance of foreign bodies into the air passages or alimentary tract. (a) All dentures should be removed. (b) The mouth should be inspected for crowns fillings and loose or weak teeth so that these may be avoided when a Mason's gag is used. If this inspection is omitted mere insertion of the sponge may prove dangerous as shown by a case where a loose incisor tooth was dislodged in this way and pushed into the trachea. (c) The jaws of the Mason's gag should be covered with rubber to prevent damage to the teeth. The gag should always be used gently and the mouth opened slowly. (d) The pharynx should be properly packed. (e) The head should not be over extended. (f) The operation should be proceeded with only when anæsthesia is tranquil.

## FOREIGN BODIES LOST IN THE MOUTH

If a loose body in the mouth slips backwards into the pharynx it may then enter either the œsophagus or the air passages. Chevalier Jackson<sup>1</sup> points out that functions of the larynx other than that of speech are generally given little consideration. He observes that phylogenetically phonation is late and articulate speech as we know it very late indeed. He describes the fundamental purpose of the larynx for the great majority of animals including man to be the protection of the air passages against the entrance of food.

Any loose object in the mouth is a potential danger. Spicules of bone broken or whole teeth fillings rubber covers from Mason's gags and vomitus have all been found in the air passages. The number of potential foreign bodies should be reduced as far as possible. Small pledgets of wool should not be used for swabbing out tooth sockets the throat sponge or pack should have attached to it a tape which hangs out of the mouth. The fact that accidents due to foreign bodies are so rare in spite of the great number of operations in the mouth is a great tribute to the skill and care of dental surgeons.

only or may extend to weeks or even months, although then there are usually paroxysms of coughing. If, after an operation on the mouth, a patient develops lung trouble which does not clear up quickly, the possibility of a foreign body being in the lungs must always be considered. A prolonged symptomless interval is often responsible for failure to make the correct diagnosis when symptoms do eventually appear. If the advice given (p. 361) has been followed and the possibility of a foreign body being in the lung has been communicated to the family doctor immediately after the operation, he will be in a position to make an early diagnosis in an otherwise baffling case. Early treatment can then be instituted and this is important since delay may prove fatal.

A septic foreign body wherever situated eventually gives rise to suppuration and abscess formation. The gravity of the condition depends to a considerable extent on the locality of the abscess and the ease with which it can be drained. In the subcutaneous tissues an abscess is at once recognisable and can be incised and drained without delay. In the lung the symptoms of an abscess vary greatly. Usually an irritating cough develops first soon to be followed by expectoration. The sputum may or may not be blood-streaked and eventually becomes foul smelling. Fever is a late sign and is usually irregular. In other cases influenza or bronchitis is liable to be diagnosed and the gravity of the condition is not realised until instead of recovering within the expected time the patient becomes worse. If the presence of a foreign body is not diagnosed until symptoms of a lung abscess have developed the patient's condition will deteriorate rapidly and death will be inevitable unless the foreign body is removed at once and adequate drainage established.

Provided treatment is not delayed, the prognosis when a foreign body is situated in the bronchus is excellent. Removal by an expert is easy particularly if performed early. Chevalier Jackson writes

The patient from whom a foreign body has been removed within a few days by peroral bronchoscopy has a hard time trying to show that he has suffered much or that he is any the worse physically. On the other hand the patient with lung suppuration due to neglect of early diagnosis and removal has a good case with which to elicit the sympathy of the jury.

## ACUTE RESPIRATORY OBSTRUCTION

This may occur in the larynx from spastic adduction of the vocal cords or from impaction of a foreign body. The treatment depends



body in the lungs is always grave unless the patient receives urgent and skilful treatment

### Foreign Bodies in the Air Passages

Even when the patient is conscious a foreign body may disappear into the air passages without producing immediate symptoms <sup>2</sup> and the chance of this occurring is of course much greater when the patient is anæsthetised. Although it is true that under light anæsthesia a foreign body is more likely to be swallowed than inspired there is no guarantee that the airway will not be invaded for the coughing which a foreign body at the larynx may incite is preceded commonly by a deep inspiration which increases the risk of its entering the larynx. As soon as anæsthesia is deep enough to abolish the cough reflex any small foreign body which is lost backwards from the mouth is likely to fall directly through the open glottis into one or other bronchus usually the right where it may not give rise to respiratory signs or difficulty. *[The fact that this accident may occur without any immediate signs must be emphasised]*

The most common causes of the entrance of a foreign body into the larynx are

1 Allowing an unconscious patient who is vomiting to remain in a position where the vomitus does not drain away at once. If the patient is supine his head should be lowered and turned to one side (fig 171 p 289) if he is in the dental chair his head should be pushed well forward (fig 82 p 233)

2 Pulling the tongue forward during the act of vomiting since this makes swallowing and closure of the larynx difficult or impossible

3 Extending the head too far in the dental chair as is liable to occur when the anæsthetist extends the head to help the dentist when he is extracting an upper wisdom tooth. The larynx cannot then be raised even voluntarily and swallowing is impossible so that the entrance to the larynx cannot be protected. The anæsthetist should impress upon himself the vulnerability of the larynx when the head is fully extended by attempting to swallow with his head in this position. Further it is difficult to pack off effectively behind the upper molar teeth. This position is illustrated in fig 181 p 296 and an instance is given on p 362 of a foreign body entering into the lungs in a case where the head was over-extended.

Subsequent events depend on the <sup>(1)</sup> size of the foreign body and where it lodges on its infectivity and on the resistance of the tissues. There is usually a symptomless interval and this may last a few days

which follow. Since the introduction of *d* tubocurarine chloride however, treatment more prompt than was previously possible is available to relieve the condition before it develops to the stage of respiratory arrest.

When nitrous oxide is given to a resistant patient, if he has not been premedicated and if supplementary anaesthesia is not used he does not become anaesthetised unless at certain stages of anaesthesia oxygen deprivation is so severe that there is a danger of laryngeal spasm. The following six cases in our own experience are typical. All six patients were plethoric and of the 'bull-necked' type and five of them were men.

At a demonstration by R. R. M. of nitrous oxide anaesthesia for dental cases a man aged 50 stout and short-necked was brought in with a history of previous difficulty with this anaesthetic administered nasally. It was stated that the man was unable to breathe through his nose and also that it had been impossible to anaesthetise him sufficiently to allow extractions to be performed. Several teeth had to be removed. The patient was asked to blow his nose and was found to be perfectly well able to breathe through it. In fact, unconsciousness was induced without even covering the patient's mouth. The anaesthetist accordingly felt that the previous administration had probably been unskilful and proceeded in the usual way. The patient was noisy and still mumbling at a time when cyanosis was so marked that one would have expected no such restlessness even in this type of individual. Administration of  $N_2O$  was continued but despite marked anoxaemia, the anaesthesia was not deep enough to prevent reflex responses and the patient reverted to mouth breathing as soon as the extraction was attempted. The operation was therefore suspended and the depth of anaesthesia still further increased here unwisely by pushing  $N_2O$ . The sequence of events was then approximately as follows. Respiration became stertorous and snatchy and during the following ten seconds inspiratory efforts were ineffective and respiration ceased. Although from the stertor and from the patient's appearance it was obvious that there was marked anoxaemia, the cessation of respiration was in view of the muscular activity 30 seconds previously unexpectedly sudden. The tongue at this stage had become small and spastic, and almost wooden in consistency. A few seconds later it relaxed like the rest of the body musculature and became large and flabby. The pupil became widely dilated and the eye death-like. Since artificial respiration in the chair was found to be ineffective the patient was placed on the floor where it was continued. Ventilation was impossible since air could neither enter nor leave the

on which of these two conditions is responsible but without laryngoscopy it may be impossible to decide with certainty between these alternatives. To provide for the possibility that the obstruction is caused by a foreign body the anæsthetist should ask for the tracheotomy instruments to be set out.

### Signs of Acute Respiratory Obstruction

Even in the anæsthetised patient respiratory movements may continue for some seconds after they have ceased to be effective. During this time the indrawing of the suprasternal and supraclavicular notches constricts the veins at the root of the neck and is largely responsible for producing cyanosis and extreme congestion of the face. The heart continues to beat for some minutes after respiration has ceased and venous engorgement increases since blood is forced into the vessels at a time when its return is not aided by the pump-like mechanism of the thorax.

### Laryngeal Spasm

This condition is described on pp 298-301. In clinical anæsthesia it may result (i) at a late stage of anoxæmic anoxia from an ill-advised attempt to subdue an unpremedicated anæsthetic resistant patient with nitrous oxide (ii) from attempts to intubate the uncocainised larynx under *light* barbiturate or *light* cyclopropane anæsthesia in either of which laryngeal spasm is extremely probable.

In all these cases the respiratory centre is depressed and the spasm may cause acute respiratory obstruction persisting until the patient is moribund. The alarming appearance of the patient and the fact that artificial respiration is at first unsuccessful (p 311) are likely to tempt the anæsthetist to perform tracheotomy under the erroneous impression that *respiration is obstructed by a foreign body*. With proper treatment the patient's condition undeniably serious is however not as grave as his appearance would suggest for the very persistence of muscular tone shown by the laryngeal spasm can be regarded as in itself reassuring. Although after cessation of respiration the patient's condition steadily deteriorates, if the cardiac musculature is reasonably healthy the heart will still continue to beat for some time. When usually about one minute after respiration has ceased (it seems much longer to the anæsthetist!) the laryngeal spasm has passed off because the patient is *in extremis* the airway becomes free again and artificial respiration or inflation of the lungs with oxygen will then be effective. This is the sequence of events as illustrated in the examples

*d* tubocurarine chloride may well result in respiratory arrest from diaphragmatic paralysis. This condition, however, is easily dealt with by inflation which becomes possible as soon as the laryngeal spasm is abolished by the *d* tubocurarine chloride. Inflation must be maintained until normal respiration begins once more, when the *d*-tubocurarine chloride is eliminated.

The effect of administration of *d* tubocurarine chloride is to reduce the time during which inflation is impossible because of spasm thus ensuring prompt oxygenation and avoiding strain on the cardiovascular mechanism. The essential treatment is, as it always was, to get oxygen into the lungs as soon as possible.

Laborde's method of intermittent traction on the tongue<sup>4</sup> will restore respiration only if the reflex mechanisms on which it depends are still effective. The tongue is grasped by forceps or held through a towel and drawn well out of the mouth and then released. If after this cycle has been repeated seven or eight times in half a minute the patient still makes no effort to breathe artificial respiration must be instituted. The chest will be in the position of expiration and while laryngeal spasm persists artificial respiration will be ineffective, since the cords will be further approximated in the inspiratory phase (fig 191, p 300). Attempts at artificial respiration by manual methods or by inflation with oxygen must therefore be gentle, and must be continued so that as soon as the glottis opens air will enter the chest. The question whether tracheotomy should be done or not must be decided by the man on the spot. Grave as the condition of the patient is we have not heard of any case where laryngeal spasm has proved fatal because tracheotomy was not performed. Nevertheless if a laryngoscope is not available to confirm the diagnosis, early tracheotomy will probably be justifiable particularly if there is any suspicion that the obstruction is due to a foreign body.

If with the aid of a laryngoscope the anaesthetist finds there is no foreign body in the larynx he may attempt to pass a Magill's tube into the trachea. The attempt may be successful if the spasm is not extreme or is beginning to relax but vigorous efforts should not be made nor a tube of firmer consistency used on account of the possibility of injuring the vocal cords.

### Impaction of a Foreign Body

A tooth fragment or even a whole tooth rarely causes acute respiratory obstruction since they are usually small enough to pass through the glottis. Obstruction may occur however, from the impaction of a sponge or vomitus in the larynx usually just beyond

lungs entrance being prevented by laryngeal spasm and exit by the fact that the chest was already in the extreme expiratory position. Tongue traction and artificial respiration were continued and about a minute later the laryngeal spasm ceased and ventilation became effective. Normal respiration then began quickly and the patient was replaced in the chair. In spite of the extreme depression to which he had been reduced the patient promptly became restless again. The attempt to anæsthetise this patient with  $N_2O$  alone was obviously ill advised. ethyl chloride was accordingly used to supplement the  $N_2O$ . the operation was successfully concluded and the patient walked away none the worse for his experience.

Of the other five patients three were in the dental chair and the other two on the operating table. The three former were anæsthetised by students in the out patients department. Anoxæmia was marked and laryngeal spasm developed and made ventilation impossible. As artificial respiration in the chair was ineffective the patients were in each case placed on the floor and the attempt to carry it out continued. It was difficult to believe that a foreign body was not the cause of obstruction so much so that in one case tracheotomy instruments were asked for but recovery took place before they were produced. After tongue traction and artificial respiration for about a minute air entered the chest and normal respiration was promptly resumed.

In the two cases on the operating table the laryngeal spasm was verified with a laryngoscope and it was found impossible to pass a Magill's tube. When the cords were seen to have relaxed the tube passed without difficulty and the lungs were easily inflated with oxygen. Subsequent anæsthesia and recovery in these two cases also were uneventful.

The following case illustrates the production of severe laryngeal spasm which is especially liable to occur under light barbiturate narcosis.

A colleague who tried for some time to pass an endotracheal tube under light hexobarbitone narcosis found that laryngeal spasm occurred and persisted even after the attempt had been abandoned. The patient became cyanosed and the spasm passed off and artificial respiration became effective only after his condition had further deteriorated as shown by cardiac irregularity.

**Treatment**—An effective way of dealing with laryngeal spasm is the prompt intravenous administration of *d* tubocurarine chloride in a full curarising dose. About 15 mg should be given as soon as the condition is recognised but this should be done only if adequate means of inflating the lungs with oxygen are at hand since the

*d* tubocurarine chloride may well result in respiratory arrest from diaphragmatic paralysis. This condition however, is easily dealt with by inflation which becomes possible as soon as the laryngeal spasm is abolished by the *d* tubocurarine chloride. Inflation must be maintained until normal respiration begins once more when the *d* tubocurarine chloride is eliminated.

The effect of administration of *d* tubocurarine chloride is to reduce the time during which inflation is impossible because of spasm, thus ensuring prompt oxygenation and avoiding strain on the cardiovascular mechanism. The essential treatment is, as it always was, to get oxygen into the lungs as soon as possible

Laborde's method of intermittent traction on the tongue<sup>4</sup> will restore respiration only if the reflex mechanisms on which it depends are still effective. The tongue is grasped by forceps or held through a towel and drawn well out of the mouth and then released. If after this cycle has been repeated seven or eight times in half a minute the patient still makes no effort to breathe artificial respiration must be instituted. The chest will be in the position of expiration and while laryngeal spasm persists artificial respiration will be ineffective since the cords will be further approximated in the inspiratory phase (fig 191, p 300). Attempts at artificial respiration by manual methods or by inflation with oxygen must therefore be gentle and must be continued so that as soon as the glottis opens air will enter the chest. The question whether tracheotomy should be done or not must be decided by the man on the spot. Grave as the condition of the patient is, we have not heard of any case where laryngeal spasm has proved fatal because tracheotomy was not performed. Nevertheless, if a laryngoscope is not available to confirm the diagnosis, early tracheotomy will probably be justifiable, particularly if there is any suspicion that the obstruction is due to a foreign body.

If with the aid of a laryngoscope the anaesthetist finds there is no foreign body in the larynx he may attempt to pass a Magill's tube into the trachea. The attempt may be successful if the spasm is not extreme or is beginning to relax but vigorous efforts should not be made nor a tube of firmer consistency used on account of the possibility of injuring the vocal cords.

### Impaction of a Foreign Body

A tooth fragment or even a whole tooth rarely causes acute respiratory obstruction since they are usually small enough to pass through the glottis. Obstruction may occur however from the impaction of a sponge or vomitus in the larynx usually just beyond

reach of an exploring finger and in such a position that if it cannot be extracted by forceps tracheotomy will relieve the condition

A sponge or mouth pack is potentially very dangerous. When the fresh sponge is placed in the normal position before the operation is commenced it is elastic and big enough to be an effective pack, and it is difficult to believe that it could enter the larynx. It should however be borne in mind that during an operation it often becomes sodden with blood and mucus and is then easily compressed into a small slippery mass. Ceasing to be a protection it now becomes a menace since it can be easily pushed backwards behind the base of the tongue and thence sucked into the larynx.

Some years ago one of us was anæsthetising a patient in the dental chair for the extraction of a lower 6 which proved more difficult than was expected. During the long operation the patient salivated profusely was slightly restless and the noise made by the expiratory valve suddenly stopped. Respiratory efforts continued but were ineffective. Investigation showed that the sponge had disappeared from sight although it could be touched with the tip of the finger just behind the epiglottis. It was however too far back to be gripped except by the forceps with which it was successfully removed. Inspection showed that the sponge originally the size of an orange had shrunk to the size of a walnut.

Since this alarming experience we have made it an invariable rule that any sponge or mouth pack should have attached to it a tape the end of which hangs out of the mouth.

Very rarely the obstruction is subglottic. Blood clot has been known to slip through the glottis and cause acute respiratory obstruction by blocking the bifurcation of the trachea. In such a case tracheotomy will afford no relief unless forceps are passed through the wound and the clot withdrawn.

**Treatment**—If a laryngoscope is available the foreign body should be removed with forceps under direct vision care being taken not to push it farther into the larynx. If a laryngoscope is not to hand an attempt should be made to remove the obstruction by the fingers or by a pair of forceps.

If these efforts are not successful and if anæsthesia is still light the patient may be given one good thump on the back in the hope of making him cough and expel the foreign body. If a child the patient may at the same time be held upside down.

For cases under nitrous oxide anæsthesia where a foreign body obstructs the larynx and cannot be removed digitally or by forceps

McKesson recommended that a mask should be applied firmly to the face and oxygen delivered under pressure. The rationale of the method is that if the laryngeal opening is not completely occluded, oxygen will be forced through any chink. Since recovery from  $N_2O$  is quick the patient will soon regain his cough reflex and may be able to expel the foreign body. We have no first hand information of any case where oxygen has been thus employed and it is obvious that there is some danger of pushing the foreign body through the glottis. In any case the method is unsuitable if a more potent anæsthetic than  $N_2O$  is being used since then oxygen will not lighten anæsthesia sufficiently quickly.

If these attempts are unsuccessful, *the trachea (or in exceptional circumstances the larynx) must be opened at once*.

The indication for making an artificial opening into the air passages is the occurrence of acute respiratory obstruction caused by a foreign body at the inlet of the larynx and by reason of which the patient is in extremis. The opening can be made into the trachea (tracheotomy) or into the larynx (laryngotomy). The former is the more complicated but it is the operation of choice. The more simple horizontal incision of laryngotomy will save life, and the operator should not hesitate to undertake this operation if he feels his knowledge is not enough to enable him to perform tracheotomy. The disadvantage of laryngotomy is the liability to subsequent stenosis.<sup>5</sup>

## EMERGENCY TRACHEOTOMY

In theory tracheotomy is a simple operation easily performed by the student in his surgical anatomy classes but it presents great difficulties in actual practice because of the circumstances in which it has to be done. The dramatic suddenness with which the condition develops makes a certain amount of flurry inevitable. by the time the operation is begun the patient is deeply congested, so that the field of operation is obscured by profuse venous hæmorrhage and when emergency tracheotomy has to be performed in a dental surgery the instruments of choice and skilled assistance are rarely available.

If the standard instruments are not available the operator can using only a knife make an adequate opening into the air passages. The trachea can be opened within 60 seconds after locating the site of incision and the operation should not take more than two minutes if the operator is familiar with the salient features of the procedure.

*Instruments Required* — A tracheotomy set containing the instruments listed and illustrated below is part of the equipment of every operating theatre and should be available in every well appointed dental surgery.



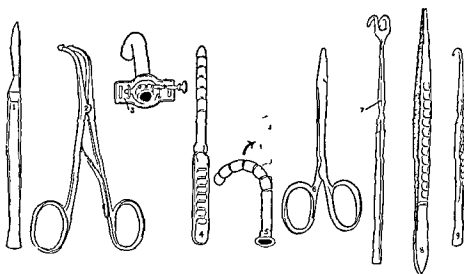


FIG 195

- ✓ 1 Scalpel
- ✓ 2 Tracheal dilator
- ✓ 3 Tracheotomy tube
- ✓ 4 Introducer
- ✓ 5 Inner tube

- ✓ 6 Several pairs of artery forceps
- ✓ 7 Retractors
- ✓ 8 Dissecting (conveying) forceps
- ✓ 9 Tracheal hook

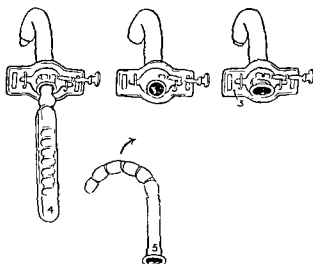


FIG 196

The introducer (4) fits into the tracheotomy tube (3) and is used to guide it into position

When the tube is in position and the introducer has been withdrawn the flexible inner tube (5) is inserted

## Anatomy

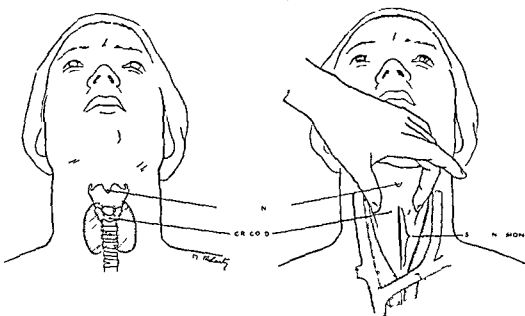


FIG 197

The trachea and its immediate relations are shown in relation to more superficial structures in the neck. The great v.e.s.s. lie deep to the sternomastoid muscles. The oesophagus lies immediately behind the trachea.

If the head is kept strictly in the midline, the danger of injuring the great vessels is very remote and exists only in the rare cases in which in children the left innominate vein crosses to the right side above the level of the manubrium sterni. The landmarks employed and the site of the incision are shown. The cricoid cartilage is more easily identified than is generally supposed. The thumb and middle finger of the operator's left hand are placed on either side of this in the depression between the sternomastoid muscles and the trachea pushing the former laterally. The trachea is steadied and rendered prominent by this manoeuvre.

The incision to be made in the trachea is vertical in the midline upward, through the third and second rings. Almost invariably this necessitates incising the isthmus of the thyroid gland. Fig 198 shows also the site of a laryngotomy incision, a transverse slit through the cricothyroid membrane.

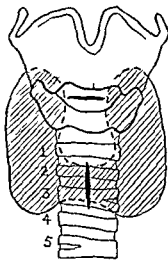


FIG 198

### The Operation

The essential points of this operation are as follows the landmarks are the cricoid cartilage and the midline of the neck the midline is strictly adhered to bleeding is disregarded the thumb and middle finger of the left hand once they have identified and grasped the cricoid cartilage retain this position throughout the operation the left index finger may be used to explore the upper part of the wound the cricoid and first ring of the trachea must not be incised the incision in the trachea is vertical and upwards through the third and second rings, care being taken not to incise too deeply for fear of injuring the posterior wall of the trachea and the œsophagus, once the tracheal incision is made it should be kept spread until an appropriate tracheotomy tube is inserted bleeding is attended to only after respiration is restored

The steps in the operation are illustrated in the following diagrams For teaching purposes the structures encountered have been clearly shown but it must be remembered that in actual fact in emergency tracheotomy very little is seen except blood and that structures can be identified only by palpation Bleeding is a prominent feature of the operation and is due largely to engorgement of the tissues caused by resistance to the venous return to the thorax The best way to stop this bleeding is to restore the airway when resumption of respiratory movements will reduce the greatly raised venous pressure to normal Not until respiration is restored are bleeding points dealt with by applying artery forceps and ligatures

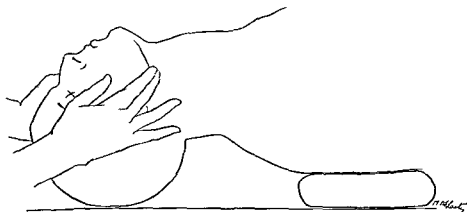


FIG 199

The patient is placed on his back with a cushion or folded coat under the shoulders to extend the head and stretch the neck thus

bringing the trachea as near the surface as possible The object of this position is to make the trachea accessible and, equally important to fix it Even if there is only one assistant, his first duty is to maintain the head firmly in this position

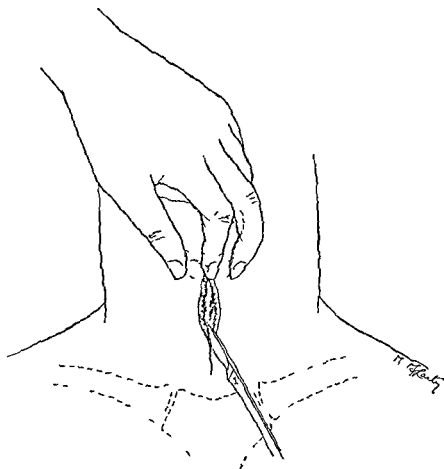


FIG 200

If time allows the throat is painted with spirit or iodine The cricoid is localised with the fingers of the left hand, thumb to the right and middle finger to the left the index finger resting on the midpoint The index finger is lifted momentarily and the skin incision made in the midline from the middle of the cricothyroid membrane to a finger's breadth above the sternal notch

### The Operation

The essential points of this operation are as follows the landmarks are the cricoid cartilage and the midline of the neck the midline is strictly adhered to bleeding is disregarded the thumb and middle finger of the left hand, once they have identified and grasped the cricoid cartilage retain this position throughout the operation the left index finger may be used to explore the upper part of the wound the cricoid and first ring of the trachea must not be incised the incision in the trachea is vertical and upwards through the third and second rings, care being taken not to incise too deeply for fear of injuring the posterior wall of the trachea and the œsophagus once the tracheal incision is made it should be kept spread until an appropriate tracheotomy tube is inserted bleeding is attended to only after respiration is restored

The steps in the operation are illustrated in the following diagrams For teaching purposes the structures encountered have been clearly shown but it must be remembered that in actual fact in emergency tracheotomy very little is seen except blood and that structures can be identified only by palpation Bleeding is a prominent feature of the operation and is due largely to engorgement of the tissues caused by resistance to the venous return to the thorax The best way to stop this bleeding is to restore the airway when resumption of respiratory movements will reduce the greatly raised venous pressure to normal Not until respiration is restored are bleeding points dealt with by applying artery forceps and ligatures

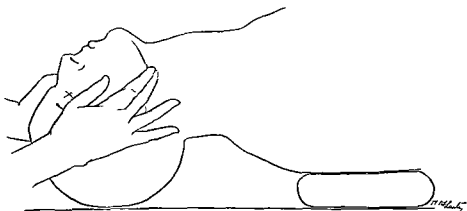


FIG 199

The patient is placed on his back with a cushion or folded coat under the shoulders to extend the head and stretch the neck thus

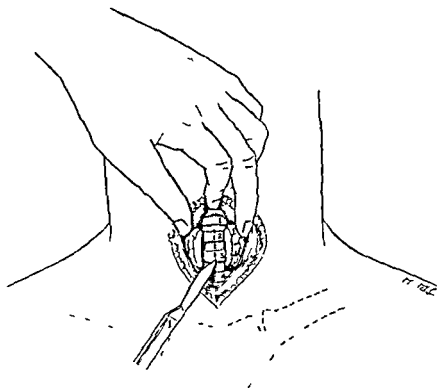


FIG 202

Under these conditions the trachea is not always easy to find, but the palpating finger should not fail to recognise the cartilaginous rings once they are encountered. The thumb and middle finger of the left hand clear the field by pushing sideways the cut thyroid gland and the sternohyoid and sternothyroid muscles. The knife is held with its back towards the sternal notch. Excess of blood is mopped from the operation field and the scalpel facing upwards, is inserted into the trachea. Since the cartilaginous rings of the trachea are deficient in its posterior aspect, great care must be taken not to incise too deeply for fear of injuring the œsophagus. The incision is made vertically upwards through the third and second rings of the trachea. The first ring of the trachea and the cricoid cartilage are diligently avoided.

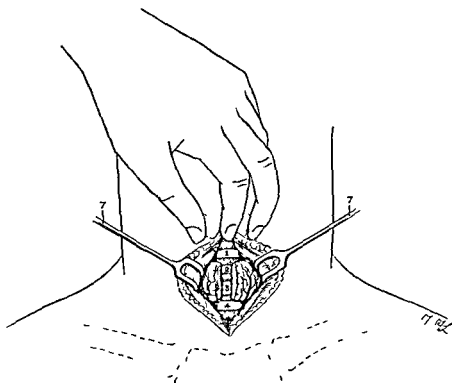


FIG 201

[The tip of the index finger is now inserted into the upper part of the wound defining the lower border of the cricoid cartilage. The upper part of the incision is deepened down to the trachea. No attempt is made to avoid the isthmus of the thyroid gland which in fact is usually divided. Bleeding is profuse and is disregarded until the trachea is defined. If a second assistant is available the wound edges may be held apart by retractors.]

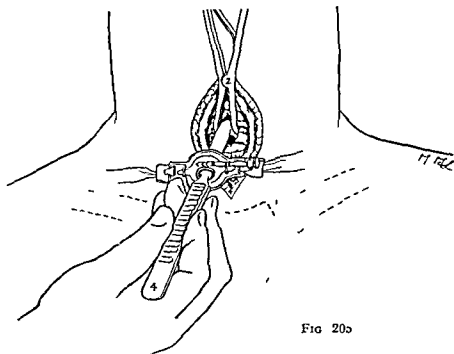


FIG 205

The introduction of the tube is often the most difficult part of the operation and must be done with deliberation. It is surprisingly easy to introduce it into the soft tissues beside or in front of the trachea.

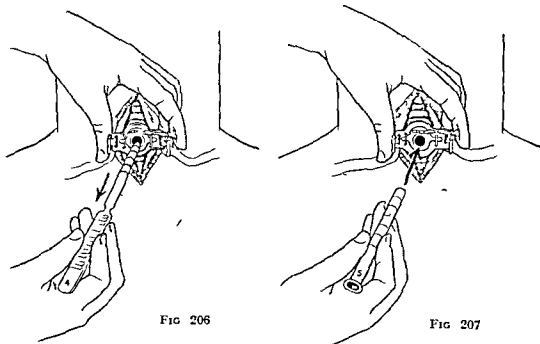


FIG 206

FIG 207

Once the tracheostomy tube is in position the introducer is immediately removed and the inner tube inserted. If respiratory effort has



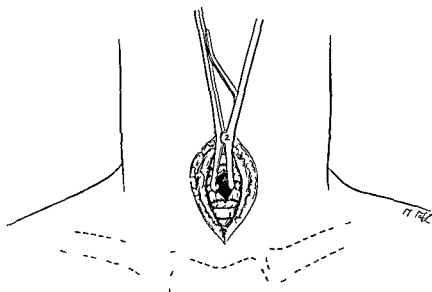


FIG 203

The wound is quickly mopped as free of blood as possible to prevent the blood being inhaled and the tracheal incision spread with the tracheal dilator (which is kept in position until the tracheotomy tube is inserted)

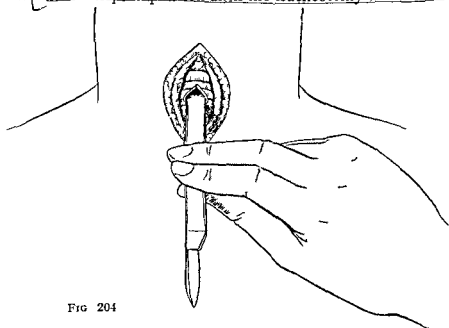


FIG 204

If a tracheal dilator is not available the opening into the trachea can be made patent by inserting the handle of the scalpel and turning it through a right angle

thyroid cartilage which rests upon it is incomplete posteriorly. The risk of stenosis is practically non-existent if the incision is made below the first ring of the trachea as described. If, however, the first ring of the trachea and more particularly the cricoid cartilage the only *complete* ring of cartilage in the respiratory tract, is incised, there is grave danger of subsequent cicatricial stenosis of the air passage sufficient to cause chronic respiratory obstruction. If the emergency operation has involved the cricoid cartilage or first ring of the trachea, the expert under whose charge the patient is placed may see fit at once to perform a subsequent tracheotomy lower down, thus diminishing the risk of stenosis. A second operation might also be performed for the same reason after an emergency laryngotomy.

### After-care

Care must be taken that the tube does not become blocked by secretions. The inner tube should be changed as often as necessary, and in any case every 3-4 hours. The outer tube should be changed daily, to prevent tracheal ulceration and secondary hæmorrhage. A layer of gauze is kept over the opening of the tube to prevent inhalation of foreign bodies. Cough sedatives and narcotics should not be employed, since the cough reflex is an important protective mechanism in preventing the accumulation of bronchial secretion.

The tracheotomy tube can be removed when the condition for which it was inserted is completely relieved. This is usually the case in two to three days when obstruction was due to impaction of a foreign body.

## ORDERLY TRACHEOTOMY

This is performed for relief of respiratory obstruction of a less acute nature than that necessitating emergency tracheotomy and is generally carried out under local anæsthesia. The principles are the same as for emergency tracheotomy the main difference being that hæmostasis is effected as the operation proceeds. The isthmus of the thyroid is divided between clamps the tracheal incision then made as described and the tracheotomy tube inserted.

## LARYNGOTOMY

The larynx extends to the lower border of the cricoid cartilage. Laryngotomy consists in making a transverse opening through the

ceased artificial respiration must be instituted Not until respiration is restored is hæmostasis effected Artery forceps are omitted from the diagram for the sake of clarity

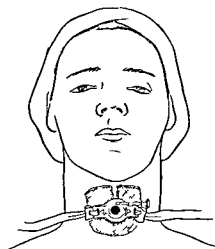


FIG 208

Gauze is packed between the skin and the tracheotomy tube which is then secured by tape round the neck (fig 208) A layer of gauze can be placed in front of the opening of the cannula to prevent any foreign body from being inhaled Breathing should now be effortless

These diagrams do not show the use of the dissecting forceps and the tracheal hook illustrated in the tracheotomy set The former are of general use in any surgical operation The tracheal hook is classically used for insertion under the lower border of the cricoid cartilage to assist in fixing the trachea

### Removal of the Foreign Body

The stimulus of opening the trachea often provokes a bout of coughing which may expel the foreign body In any case the tracheotomy tube should be inserted and left in position since any trauma caused by the foreign body may be followed a few hours later by œdema of the glottis

If the foreign body is not expelled no attempt need be made to remove it the immediate danger having been overcome its extraction by endoscopic methods can be deferred until later

### Dangers of Tracheotomy

The great vessels of the neck may be incised unless the operator keeps strictly to the midline The œsophagus may be opened if the incision is made too deeply When this happens the subsequent infection is extremely virulent and almost invariably fatal The left innominate vein may be injured in a child if the incision is carried too low

The remote danger of tracheotomy is that of permanent laryngeal or tracheal stenosis The strongest factor in maintaining the laryngeal lumen is the cricoid cartilage which is a complete ring whereas the

## OVERDOSE OF ANÆSTHETIC

Respiratory arrest occurs when an overdose of any anæsthetic or of any muscle relaxant drug (p 195) is given. Except occasionally with chloroform, it occurs before cardiac failure, so that in the case of *inhalation* anæsthesia it is generally possible by starting artificial respiration promptly to keep the tissues oxygenated until the excess of anæsthetic has been excreted. Treatment of respiratory arrest produced by anæsthetics given by other routes is less liable to be effective (p 16).

The immediate treatment of overdose, whatever the anæsthetic or its route of administration, is the same. In this emergency the anæsthetist must rely on his hands and not on drugs. Artificial respiration is worth all the drugs in the pharmacopœia. If normal breathing cannot be restored in this way it is in our opinion highly improbable that any other method would have been successful. The beneficial effects of  $\text{CO}_2$  and of analeptics are in some cases dramatic but experimental evidence suggests that this happens only in cases which would have fared equally well or even better in the long run with oxygen alone and that for a patient *in extremis*  $\text{CO}_2$  or analeptics may actually turn the scale against him.

## ARTIFICIAL RESPIRATION

In cases where an overdose of the anæsthetic has been given or in cases of advanced oxygen lack the respiratory centre becomes depressed and respiration ceases before the heart stops beating. As long as circulation continues life can be maintained by artificial respiration. Provided irreparable damage to the vital centres has not been caused the respiratory centre will recover after sufficient anæsthetic has been excreted.

The essential feature of all methods of artificial respiration is alternating distension and deflation of the lungs. Besides supplying oxygen and removing  $\text{CO}_2$  these movements may initiate the reflexes which function in normal respiration (p 50). In all cases the airway must be kept clear and if necessary the back of the throat should be swabbed free of mucus and blood.

### Inflation with Oxygen

Some means of inflating with oxygen should be available in every operating theatre. The McKesson anæsthetic apparatus is excellent for this purpose and as an alternative the Oxford Inflator\* will be found inexpensive, compact and easy to work.

cricothyroid membrane, i.e. at a level just below the vocal cords This emergency operation is comparatively easy to perform but healing of the wound may be followed by cicatrisation causing the serious complication of laryngeal stenosis. If an operator in an emergency doubts his ability to perform a tracheotomy he would be justified in performing a laryngotomy. The patient should be transferred as soon as possible to the care of a laryngologist who would then if necessary perform a tracheotomy in order to allow the laryngotomy wound to heal early so diminishing the risk of laryngeal stenosis.

The landmarks for laryngotomy are easily recognisable. The patient is placed in the same position as for tracheotomy. A transverse skin incision between the cricoid and thyroid cartilages is deepened in the centre through the cricothyroid membrane (fig 198 p 317).

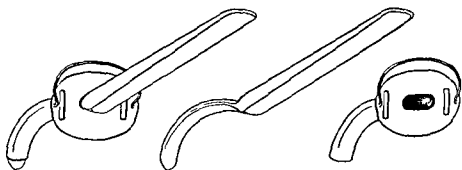


Fig 209 —Laryngotomy tube and introducer

The opening is spread with a dilator or artery forceps and a laryngotomy tube inserted. The introducer is withdrawn and the tube fixed in position by tying round the neck the tapes which are attached to the eyelets. The cross sections of a laryngotomy and a tracheotomy tube differ characteristically, the former is flat, the latter more rounded. For a given patient the appropriate laryngotomy tube is much shorter than the corresponding tracheotomy tube. An inner tube is not necessary.

The after-care required by a patient with a laryngotomy is the same as for one with a tracheotomy. It should be noted that it is unwise to leave a laryngotomy tube in for a long period since this greatly increases the danger of stenosis.

with a face piece and rebreathing bag can be used. The mask is applied firmly to the face the expiratory valve is screwed down, and the bag filled with oxygen. The anesthetist by squeezing the bag (fig 212) can produce a pressure of 10-15 mm. Hg, easily sufficient to inflate the lungs provided the airway is clear. The lungs deflate when the mask is raised from the face.

### Manual Methods of Artificial Respiration

Sometimes particularly in children respiration can be restarted when the patient is in the dental chair simply by compressing the chest once or twice and allowing it to recoil. If this is ineffective the more classical methods must be resorted to. In no case should the force applied be violent. numerous cases are on record where at post-mortems ribs have been found broken. Intermittent steady yet gentle pressure on the chest wall will be found sufficient to effect a good exchange of air. The temptation to try to hasten recovery by compressing and releasing the chest too frequently must be resisted. The optimum rate is about 12-14 per minute. The ventilation is not being effective unless the entrance and exit of air can be heard.

Schaefer's method—This is an efficient method of artificial respiration and is not fatiguing to the operator. The air exchange is

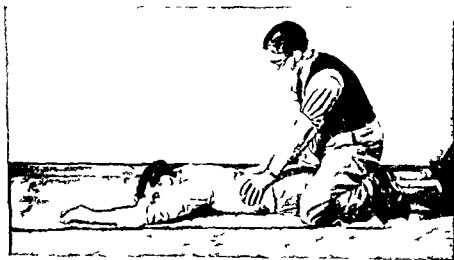


FIG 213

approximately normal. The patient lies face downwards a position which has the advantage of ensuring that the tongue falls away from the posterior pharyngeal wall and in cases of drowning that water will run out of the mouth. For these reasons it is the method of choice

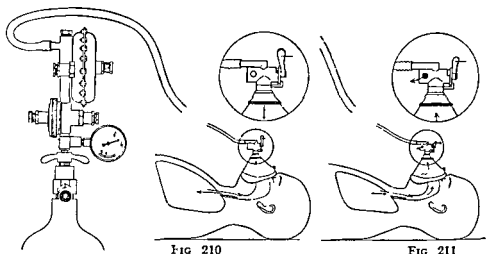


FIG 210

FIG 211

The Oxford Inflator is connected to the top of a cylinder of oxygen. By means of reducing valves the pressure of the oxygen delivered is reduced to 40 mm Hg. The face piece is applied tightly and the jaw is kept forward so as to maintain a clear airway. When the lever X is turned into the position illustrated in fig 210 the lungs are inflated with oxygen.

When the lever is turned to the off position (fig 211) it cuts off communication with the cylinder, uncovers a hole in the mask, and allows the lungs to deflate.

If an apparatus of the inflator type is not available any apparatus

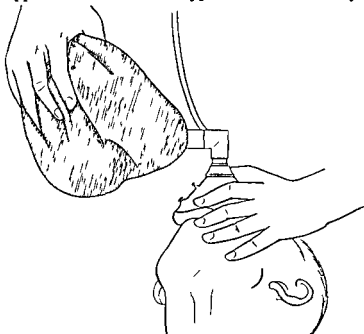


FIG 212

with a face piece and rebreathing bag can be used. The mask is applied firmly to the face, the expiratory valve is screwed down, and the bag filled with oxygen. The anesthetist by squeezing the bag (fig 212) can produce a pressure of 10-15 mm. Hg easily sufficient to inflate the lungs provided the airway is clear. The lungs deflate when the mask is raised from the face.

### Manual Methods of Artificial Respiration

Sometimes particularly in children respiration can be restarted when the patient is in the dental chair simply by compressing the chest once or twice and allowing it to recoil. If this is ineffective the more classical methods must be resorted to. In no case should the force applied be violent. Numerous cases are on record where at post-mortems ribs have been found broken. Intermittent steady yet gentle pressure on the chest wall will be found sufficient to effect a good exchange of air. The temptation to try to hasten recovery by compressing and releasing the chest too frequently must be resisted. The optimum rate is about 12-14 per minute. The ventilation is not being effective unless the entrance and exit of air can be heard.

Schaefer's method—This is an efficient method of artificial respiration and is not fatiguing to the operator. The air-exchange is

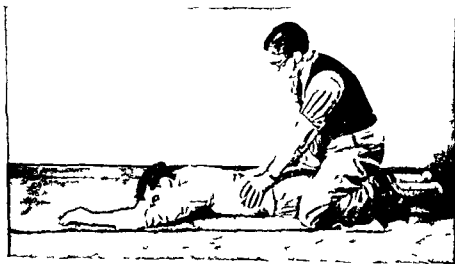


FIG 213

approximately normal. The patient lies face downwards a position which has the advantage of ensuring that the tongue falls away from the posterior pharyngeal wall and in cases of drowning that water will run out of the mouth. For these reasons it is the method of choice.



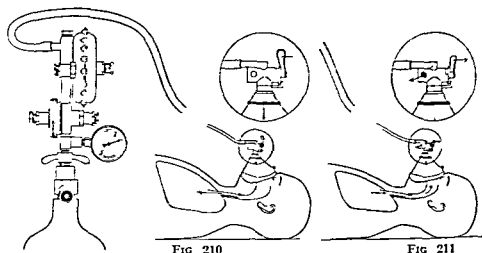


FIG 210

FIG 211

The Oxford Inflator is connected to the top of a cylinder of oxygen. By means of reducing valves the pressure of the oxygen delivered is reduced to 40 mm Hg. The face-piece is applied tightly and the jaw is kept forward so as to maintain a clear airway. When the lever  $\Lambda$  is turned into the position illustrated in fig 210 the lungs are inflated with oxygen.

When the lever is turned to the off position (fig 211) it cuts off communication with the cylinder, uncovers a hole in the mask, and allows the lungs to deflate.

If an apparatus of the inflator type is not available any apparatus

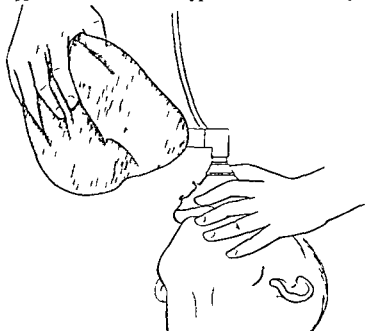


FIG 212

with a face piece and rebreathing bag can be used. The mask is applied firmly to the face, the expiratory valve is screwed down and the bag filled with oxygen. The anesthetist by squeezing the bag (fig 212) can produce a pressure of 10-15 mm. Hg, easily sufficient to inflate the lungs provided the airway is clear. The lungs deflate when the mask is raised from the face.

### Manual Methods of Artificial Respiration

Sometimes particularly in children, respiration can be restarted when the patient is in the dental chair simply by compressing the chest once or twice and allowing it to recoil. If this is ineffective the more classical methods must be resorted to. In no case should the force applied be violent. Numerous cases are on record where at post-mortems ribs have been found broken. Intermittent steady yet gentle pressure on the chest wall will be found sufficient to effect a good exchange of air. The temptation to try to hasten recovery by compressing and releasing the chest too frequently must be resisted. The optimum rate is about 12-14 per minute. The ventilation is not being effective unless the entrance and exit of air can be heard.

Schaefer's method—This is an efficient method of artificial respiration and is not fatiguing to the operator. The air exchange is

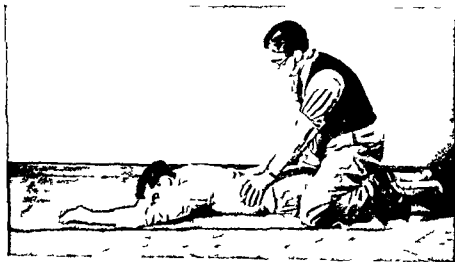


FIG 213

approximately normal. The patient lies face downwards, a position which has the advantage of ensuring that the tongue falls away from the posterior pharyngeal wall and in cases of drowning that water will run out of the mouth. For these reasons it is the method of choice

for an operator working single-handed. It is, however, usually impracticable on the operating table, and in any case is generally unnecessary since an assistant is available to keep the airway clear.

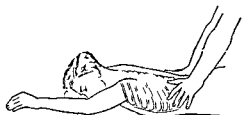


FIG 214 (After Drinker?)

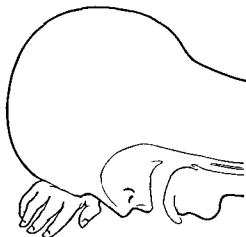


FIG 215

The patient is prone, one arm is extended forwards and the other bent at the elbow so that the head may rest on it. The head is turned to one side, the tongue falls forwards and the nose and mouth are free. The operator kneels astride the patient bearing his weight entirely upon his knees (fig 213). The ball of the thumb rests on the small of the back and the two thumbs are almost parallel and about 3 inches apart. The fingers are outstretched and the palms of the hands spread comfortably over the lowermost ribs. The arms are straight but relaxed.

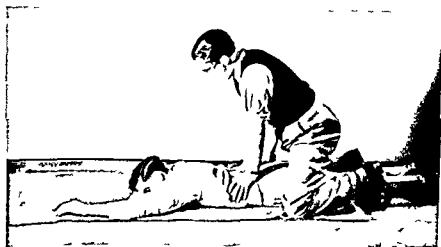


FIG 216

Expiration is produced by compressing the thorax. The operator begins by leaning forward gradually with arms kept straight until

his weight is equally distributed between his hands and knees (fig 216) In this position his arms will be vertical This part of the procedure occupies about two seconds The operator then swings back again taking all his weight on to his knees, and remains in this position for about 2 seconds to allow elastic recoil of the chest wall and abdomen to produce inspiration

Silvester's method — Inspiration is produced by extending the patient's arms above his head thereby expanding the thorax

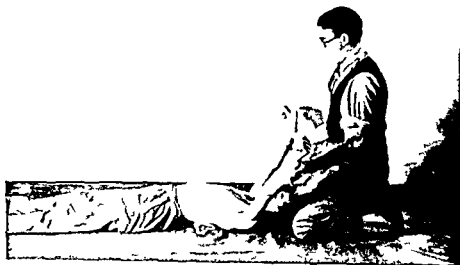


FIG 217

Expiration results from bringing the patient's arms forwards and pressing them gently but firmly against his thorax



FIG 218

Mechanically this is a poor method since the ventilation is only about one-half to two thirds of the normal

It has the further disadvantages that since the patient is on his back the tongue if not held forward by an assistant will gravitate backwards and occlude the airway (fig 219) and there is no drainage

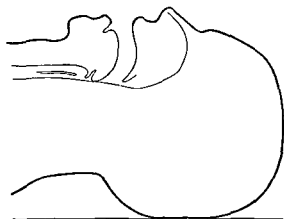


FIG 219

### CARDIAC FAILURE

In anæsthetic emergencies except occasionally in the case of chloroform poisoning the heart will continue to beat for some time after respiration has ceased a time which will vary with the causation of the respiratory arrest and with the state of the patient's heart. If the heart stops the question whether it can be made to beat again depends on the same two factors. Animal experiments suggest that after a cessation of beating there is increased irritability and contractility of the heart lasting for about 1½ minutes. During this time appropriate stimulation is often effective though it becomes less and less so as the condition of the heart deteriorates. Cases are however, on record of the heart beat being resumed even after 15 minutes.

If the circulation has been in abeyance for longer than 5 minutes the chances of the patient surviving for more than a few hours are negligible owing to the damage done to the central nervous system particularly to the respiratory centre by the long anoxia. If however, beating of the heart is restored within one or two minutes complete recovery is probable.

**Treatment** —(i) To ensure that the vital centres shall get a maximal benefit from any feeble circulation which may be present the head is lowered and if possible the patient is put into a slight Trendelenburg position. Artificial respiration preferably by inflation of the lungs with oxygen is kept up throughout.

(ii) The muscle of the right auricle is stimulated by the prick of a needle in the hope that it will contract and the beat be propagated to the ventricle



FIG 220

*Heuer's cardiac puncture needle* 5 inches long is inserted through the 3rd right intercostal space close to the sternum, the point being directed backwards and slightly towards the midline, through the wall of the right auricle. In adults the needle should be inserted  $3\frac{1}{2}$ –4 inches

(iii) If this does not start the heart beat the needle is withdrawn and an attempt is made to inject adrenaline (1 : 1 000)  $M_v-x$  into the cavity of the left ventricle which is admittedly difficult to locate. James<sup>8</sup> suggests that this should be done as follows: a point is taken at the junction of the lower  $\frac{1}{3}$ rd and upper  $\frac{2}{3}$ rd of a line joining the sternal notch and the xiphisternum. From here a line is drawn horizontally to the left midaxillary line. At the junction of the outer  $\frac{1}{3}$ rd and inner  $\frac{2}{3}$ rd of this line a straight needle is inserted for about 4 inches at right angles to the tangent on the chest wall. The fact that this procedure has been known to restart the heart beat is explicable on the assumption that although no cardiac impulse is discernible there is still a feeble circulation which carries the adrenaline into the coronary arteries.

(iv) As a final resort the hand is introduced into the abdominal cavity through an incision in the abdominal wall and the heart massaged. There is no need to incise the diaphragm since this thin muscle now completely relaxed does not prevent the heart from being grasped and kneaded against the anterior thoracic wall.

#### REFERENCES

- (1) Jackson C and Jackson C L 1937 *The Larynx and its Diseases* Philadelphia and London 17
- (2) McCrae T 1924 *Lancet* 1, 735
- (3) Jackson C 1927 *J Amer dent Ass* 14, 1341
- (4) Laborde J B V 1892 *Bull Acad Med Paris* 28, 9 519
- (5) Jackson C 1921 *Surg Gynec Obstet* 24, 392
- (6) Macintosh R R and Pratt C L G 1939 *Lancet* 1 206
- (7) Drinker C K 1938 *Carbon Monoxide Asphyxia* London New York Toronto 169
- (8) James N R 1940 Personal communication

## CHAPTER XXX

## CYLINDER VALVES AND REDUCING VALVES

At room temperature the pressure inside a cylinder containing N O remains constant at about 650 lb per square inch whilst in a cylinder of oxygen the pressure falls gradually from 1 800 lb per square inch (pp 211 212) The rate of flow of gas from the cylinder is regulated by a valve or valves

## CYLINDER VALVES

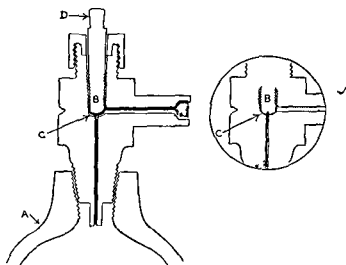


FIG 221 —Cylinder valve slightly opened Inset—valve closed

The ordinary cylinder valve fitted as standard to most cylinders does not allow of refinement in regulating the escape of gas When the cylinder is Off (circle inset) the base of the stem B fits down firmly on the valve seating C and as the cylinder is opened by turning the stem key D anti clockwise the stem is raised from the valve seating allowing gas to flow from the cylinder Delicacy of control depends partly on the accuracy of the valve seating and on the closeness of the thread of the stem

A much finer control, especially valuable where low rates of flow

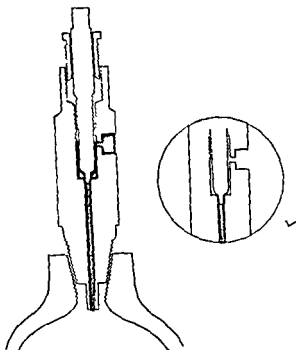


FIG. 222 — Taper valve slightly opened    Inset—valve closed

are desired is obtained if the valve has a tapered stem    Such valves are commonly referred to as taper valves

In any anæsthetic circuit a reservoir for gas at approximately atmospheric pressure is incorporated    This is necessary because if for a given patient the volume of gas or gases breathed per minute be 8 litres, the rate of flow of gas to the patient during the actual inspiratory phase will be in the neighbourhood of 20 litres per minute    The reservoir (i.e. the gas bag or the rebreathing bag) is thus depleted intermittently by inspiration and is replenished at a uniform rate from the cylinder via the valve

## REDUCING VALVES

By means of reducing valves the rate of flow of gas from the cylinder can be more delicately controlled, for in effect they render available the entire cylinder contents at a low pressure    Pressure reducing mechanisms can be employed so that gas from the cylinder is released to the patient in adequate quantities when he inspires, and the flow checked automatically when inspiration is completed    By these means economy is effected and the size of the necessary reservoir greatly reduced



## CHAPTER XXX

## CYLINDER VALVES AND REDUCING VALVES

At room temperature the pressure inside a cylinder containing  $N_2O$  remains constant at about 650 lb per square inch whilst in a cylinder of oxygen the pressure falls gradually from 1 800 lb per square inch (pp 211 212) The rate of flow of gas from the cylinder is regulated by a valve or valves

## CYLINDER VALVES

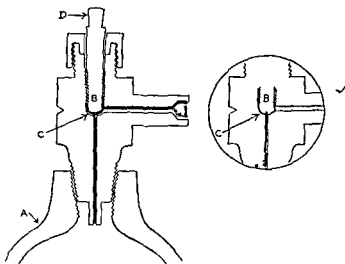


FIG. 221 —Cylinder valve slightly opened Inset—valve closed

The ordinary cylinder valve fitted as standard to most cylinders does not allow of refinement in regulating the escape of gas. When the cylinder is Off (circle inset) the base of the stem B fits down firmly on the valve seating C and as the cylinder is opened by turning the stem key D anti clockwise the stem is raised from the valve seating allowing gas to flow from the cylinder. Delicacy of control depends partly on the accuracy of the valve seating and on the closeness of the thread of the stem

the tube R into the small rubber-bag reservoir Q which is contained within the metal drum S. The outlet from the bag is obstructed by a metal disc T which exerts a pressure equivalent to about 2 mm Hg. Just before this tension is reached, the bulging bag pushes the base of the hinged rod U outwards and the head inwards forcing the flexible diaphragm V over the outlet of tube R (fig 225) thus preventing the entrance of any further gas into the bag.

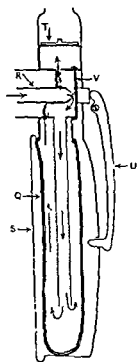


FIG 225

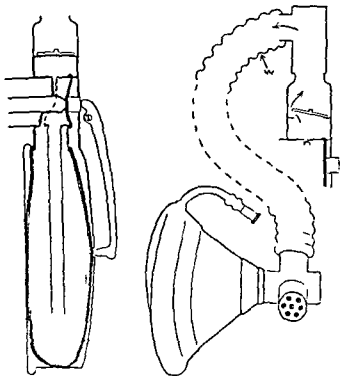


FIG 226

The reservoir is now full of gas at a pressure of just under 2 mm Hg. When the patient inspires, a negative pressure is created within the breathing tube W and the weight-valve T is raised allowing gas to enter the tube from the reservoir (figs 225-226). The bag deflates and the rod U moves with it so that the diaphragm V is no longer held against the outlet of tube R. At the end of inspiration the weight valve falls back, the bag fills and the diaphragm V is again forced back against the outlet from tube R.

**Endurance valve (British Oxygen Co)**—This valve works on a principle very similar to the McKesson.

**Adam's valve**—This valve is set by the makers so that the

In the following diagrams the reducing valve mechanisms of the Minnitt gas and air apparatus (fig 228 p 343) are illustrated

**McKesson reducing valve**—The valve has a pressure regulator G which is adjusted so that pressure is exerted on the under surface of the flexible metal diaphragm O to which plug M is attached by a steel rod. In practice it is usual for the valve regulator to be adjusted so that the tension on the spring is equivalent to a pressure of 60 lb per square inch on the diaphragm O. By this means M is displaced from the seating between the high pressure (red) and the low pressure (green) compartments so that the two compartments of the valve communicate. When the cylinder is turned on gas will now flow

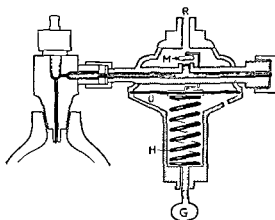


FIG 223

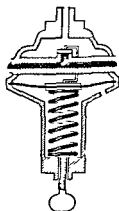


FIG 224

freely through the high pressure compartment past the valve seating into the low pressure compartment and out through R. Should the outflow through R be obstructed the pressure in the low pressure compartment will rise and as soon as it reaches 60 lb per square inch the diaphragm will be deflected as in fig 224. The plug M is now drawn on to its seating and the gas flow into the low pressure chamber is cut off. If gas is allowed to escape through R the pressure in the low pressure compartment will fall and the diaphragm O return to its previous position. Plug M is again displaced from its seating and fresh gas is admitted to the low-pressure chamber. Thus the pressure in the compartment coloured red is always that of the cylinder but the pressure in the compartment coloured green is maintained automatically at that to which the valve is adjusted—in this case 60 lb per square inch.

After the gas leaves the reducing valve proper it passes through

## CHAPTER XXXI

## ANALGESIA

THE progressive loss of sense of pain during induction is a feature which is generally overlooked and seldom put to any practical use. This phase is in fact generally passed through as quickly as possible. The loss begins early and soon reaches a stage where moderately painful stimuli are not appreciated as such although the patient still has considerable control of his faculties. Advantage can be taken of this analgesic stage to obtund fairly acute pain and yet retain the co-operation of the patient. Furthermore it interferes but little with the physiological functions, and avoids the disadvantages of prolonged anaesthesia such as would otherwise be necessary in midwifery. It is also useful in dentistry particularly where only a few cavities have to be prepared. In such cases the production of analgesia is easy, economical and characterised by lack of after-effects.

The student will do well to experience this analgesic state himself so that he can explain the phenomenon to and appreciate the feelings of a patient under similar conditions. But experiments with anaesthetics should never be performed unless a second person is present. Numerous fatalities have been reported through failure of the oxygen supply or the postponement of the removal of the mask until the capacity to do so is lost.

The anaesthetic most popular for the production of analgesia is nitrous oxide. If this is inhaled mixed with the requisite amount of air or oxygen the student will find on testing his skin with a sterile needle that while the sense of touch remains that of pain is absent. Instead of a sharp and painful prick the sensation is that associated with prodding the skin with the blunt end of a pin. Subjective symptoms vary with the individual patient. At this stage he will probably be conscious of exhilaration and a feeling of warmth throughout the body. This may be followed by lassitude and a sense of remoteness from what is going on around. Such a state is quite pleasant if the patient resigns himself to it. If on the other hand he makes an effort—inevitably unsuccessful—to keep control of his faculties he may become worried by his inability to do so.

pressure in the low-pressure compartment is maintained at about 7 lb. per square inch The pressure of gas from the cylinder forces

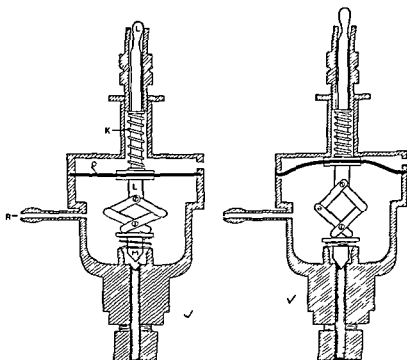


FIG 227

M from the valve seating and provided R is open gas will escape. If R is obstructed so that the pressure in the low-pressure compartment rises above 7 lb per square inch the diaphragm O will be forced upwards against the resistance of spring K. The rod L is carried upwards and the stem M attached to the lower part of the toggle (or lazy tongs) is forced down on to the valve seating preventing further gas from entering.

tions of the burr he will not be hurt. He should be told to imagine he is sitting in an armchair enjoying a rest and leaving all the work to the dentist and anaesthetist. He is not to talk, but if he feels any discomfort (the word pain is avoided) he can show this by breathing more deeply through the nose, when the situation will automatically correct itself or by signalling with his finger so that the anaesthetist may make any necessary adjustments to the mixture.

A small prop is put in the mouth and the head is tilted slightly forward, making any saliva gravitate towards the salivary ejector, which is placed under the tongue. The nose piece is held some distance off the nose and gradually lowered into position. It should never be necessary to use the mouth-cover. The patient should be talked to continuously, and, even at the expense of repetition encouraged to relax. Four or five breaths of undiluted nitrous oxide are given before air or oxygen is introduced. Air has some advantages over oxygen as a diluent for the introduction of a minute excess quantity of the latter causes a disproportionate loss of the pain reducing qualities of nitrous oxide and so makes it difficult to maintain precisely the desired level of analgesia. Except in plethoric individuals (p. 56) it is not necessary to reduce the oxygen or air intake to levels which result in cyanosis.

It is impossible to lay down any rule for determining when the desired analgesic state has been reached but it is usually attained after about four to five full breaths of nitrous oxide after which it is maintained by the addition of oxygen or air so that the nitrous oxide percentage in the mixture is in the neighbourhood of 50. A sponge should now be inserted in the mouth (not far back) with the explanation that it is being placed there to remind the patient to breathe through his nose. In point of fact its main purpose is to trap any tooth fragments or debris. Care must be taken by the operator not to catch the sponge in the revolving drill.

Analgesia gives splendid opportunity for the development of team work. The anaesthetist should signal to the dentist to begin and should tell the patient. Mr Jones is going to examine your teeth and if you feel the slightest discomfort I want you to breathe more deeply through the nose. The dentist begins on an insensitive part and gradually increases the stimulus. The dentist and anaesthetist must not speak to each other but can convey messages by signals. That a particularly sensitive part is being drilled may be betrayed by an involuntary upward twitch of the corner of the patient's mouth. This information should be communicated to the dentist as it may assist

## ANALGESIA IN DENTISTRY

The success of analgesia from the patient's point of view depends largely on gaining his confidence at the outset, and convincing him that if only he will surrender himself completely to the pleasant drowsiness he need experience neither pain nor anxiety. Once analgesia is established the anæsthetist must remain in contact with the patient maintaining his co-operation by a constant reassuring chatter. The patient is now particularly open to suggestion and the right word spoken at the right moment may make all the difference between comfortable drowsiness and an hysterical outburst. Some kind of premedication for example aspirin 10-15 gr. given to an adult one hour before operation is a valuable additional aid.

Not all patients are suitable subjects for analgesia. A certain amount of understanding is required to appreciate the existence of a state in which the sense of touch is present but not that of pain. An excitable hysterical patient may lose control and scream or laugh making operating an impossibility unless complete anæsthesia is induced. It is rare to find children trusting or co-operative enough to be suitable for analgesia.

All kinds of cavity preparations and deep scaling can be performed under analgesia. Cavities situated at the cervical margin are particularly suitable. They are generally in the front of the mouth and so are readily accessible and the debris can be washed or wiped away easily. Such cavities are often multiple and are always particularly sensitive. Analgesia allows the dentist to work rapidly so that several cavities can be prepared at one sitting. The task of dealing with cavities in the molar region is much more difficult and the danger of exposing the pulp is correspondingly greater. Access is not easy and the illumination of the back of the mouth is often unsatisfactory. The tongue or sponge may get in the way and it is difficult to keep the cavity dry and clear of debris. If a spray is used the pharynx may be irritated initiating retching or vomiting.

Analgesia will abolish pain only of a certain degree. The stimulus of severe pain may easily overcome the barrier even of deep analgesia and since the patient's mental faculties at this stage are impaired the response to this stimulus may be uncontrolled action. For this reason analgesia is unsuitable for more extractions than one or two simple ones in patients who appreciate the limitations as well as the advantages of analgesia.

The patient should be made comfortable in the chair and asked to relax. It is explained to him that whilst he will recognise the vibra-

tions of the burr he will not be hurt. He should be told to imagine he is sitting in an armchair enjoying a rest and leaving all the work to the dentist and anaesthetist. He is not to talk but if he feels any discomfort (the word 'pain' is avoided) he can show this by breathing more deeply through the nose when the situation will automatically correct itself, or by signalling with his finger so that the anaesthetist may make any necessary adjustments to the mixture.

A small prop is put in the mouth and the head is tilted slightly forward making any saliva gravitate towards the salivary ejector which is placed under the tongue. The nose piece is held some distance off the nose and gradually lowered into position. It should never be necessary to use the mouth-cover. The patient should be talked to continuously and even at the expense of repetition encouraged to relax. Four or five breaths of undiluted nitrous oxide are given before air or oxygen is introduced. Air has some advantages over oxygen as a diluent, for the introduction of a minute excess quantity of the latter causes a disproportionate loss of the pain reducing qualities of nitrous oxide and so makes it difficult to maintain precisely the desired level of analgesia. Except in plethoric individuals (p. 56) it is not necessary to reduce the oxygen or air intake to levels which result in cyanosis.

It is impossible to lay down any rule for determining when the desired analgesic state has been reached but it is usually attained after about four to five full breaths of nitrous oxide after which it is maintained by the addition of oxygen or air so that the nitrous oxide percentage in the mixture is in the neighbourhood of 50. A sponge should now be inserted in the mouth (not far back) with the explanation that it is being placed there to remind the patient to breathe through his nose. In point of fact its main purpose is to trap any tooth fragments or debris. Care must be taken by the operator not to catch the sponge in the revolving drill.

Analgesia gives splendid opportunity for the development of team work. The anaesthetist should signal to the dentist to begin and should tell the patient 'Mr Jones is going to examine your teeth and if you feel the slightest discomfort I want you to breathe more deeply through the nose.' The dentist begins on an insensitive part and gradually increases the stimulus. The dentist and anaesthetist must not speak to each other but can convey messages by signals. That a particularly sensitive part is being drilled may be betrayed by an involuntary upward twitch of the corner of the patient's mouth. This information should be communicated to the dentist as it may assist



## ANALGESIA IN DENTISTRY

The success of analgesia from the patient's point of view depends largely on gaining his confidence at the outset, and convincing him that if only he will surrender himself completely to the pleasant drowsiness he need experience neither pain nor anxiety. Once analgesia is established the anaesthetist must remain in contact with the patient maintaining his co-operation by a constant reassuring chatter. The patient is now particularly open to suggestion and the right word spoken at the right moment may make all the difference between comfortable drowsiness and an hysterical outburst. Some kind of premedication for example aspirin 10-15 gr given to an adult one hour before operation is a valuable additional aid.

Not all patients are suitable subjects for analgesia. A certain amount of understanding is required to appreciate the existence of a state in which the sense of touch is present but not that of pain. An excitable hysterical patient may lose control and scream or laugh making operating an impossibility unless complete anaesthesia is induced. It is rare to find children trusting or co-operative enough to be suitable for analgesia.

All kinds of cavity preparations and deep scaling can be performed under analgesia. Cavities situated at the cervical margin are particularly suitable. They are generally in the front of the mouth and so are readily accessible and the debris can be washed or wiped away easily. Such cavities are often multiple and are always particularly sensitive. Analgesia allows the dentist to work rapidly so that several cavities can be prepared at one sitting. The task of dealing with cavities in the molar region is much more difficult and the danger of exposing the pulp is correspondingly greater. Access is not easy and the illumination of the back of the mouth is often unsatisfactory. The tongue or sponge may get in the way and it is difficult to keep the cavity dry and clear of debris. If a spray is used the pharynx may be irritated initiating retching or vomiting.

Analgesia will abolish pain only of a certain degree. The stimulus of severe pain may easily overcome the barrier even of deep analgesia and since the patient's mental faculties at this stage are impaired the response to this stimulus may be uncontrolled action. For this reason analgesia is unsuitable for more extractions than one or two simple ones in patients who appreciate the limitations as well as the advantages of analgesia.

The patient should be made comfortable in the chair and asked to relax. It is explained to him that whilst he will recognise the vibra-

tions of the burr he will not be hurt. He should be told to imagine he is sitting in an armchair enjoying a rest and leaving all the work to the dentist and anaesthetist. He is not to talk but if he feels any discomfort (the word 'pain' is avoided) he can show this by breathing more deeply through the nose when the situation will automatically correct itself or by signalling with his finger so that the anaesthetist may make any necessary adjustments to the mixture.

A small prop is put in the mouth and the head is tilted slightly forward making any saliva gravitate towards the salivary ejector, which is placed under the tongue. The nose piece is held some distance off the nose and gradually lowered into position. It should never be necessary to use the mouth-cover. The patient should be talked to continuously and even at the expense of repetition encouraged to relax. Four or five breaths of undiluted nitrous oxide are given before air or oxygen is introduced. Air has some advantages over oxygen as a diluent for the introduction of a minute excess quantity of the latter causes a disproportionate loss of the pain reducing qualities of nitrous oxide and so makes it difficult to maintain precisely the desired level of analgesia. Except in plethoric individuals (p. 56) it is not necessary to reduce the oxygen or air intake to levels which result in cyanosis.

It is impossible to lay down any rule for determining when the desired analgesic state has been reached but it is usually attained after about four to five full breaths of nitrous oxide, after which it is maintained by the addition of oxygen or air so that the nitrous oxide percentage in the mixture is in the neighbourhood of 50. A sponge should now be inserted in the mouth (not far back) with the explanation that it is being placed there to remind the patient to breathe through his nose. In point of fact its main purpose is to trap any tooth fragments or debris. Care must be taken by the operator not to catch the sponge in the revolving drill.

Analgesia gives splendid opportunity for the development of team work. The anaesthetist should signal to the dentist to begin and should tell the patient. Mr Jones is going to examine your teeth and if you feel the slightest discomfort I want you to breathe more deeply through the nose. The dentist begins on an insensitive part and gradually increases the stimulus. The dentist and anaesthetist must not speak to each other but can convey messages by signals. That a particularly sensitive part is being drilled may be betrayed by an involuntary upward twitch of the corner of the patient's mouth. This information should be communicated to the dentist as it may assist

him in localising and avoiding the root canal. Minor variations in the depth of analgesia are of little importance provided the anæsthetist by patting the patient on the shoulder and talking to him in a simple straightforward manner keeps in touch with him almost continuously. It is easy particularly for the placid patient to slip from the state of analgesia into unconsciousness so caution must be exercised in promising a patient that he will remain awake throughout. Sometimes the whole procedure is so comfortable for the patient that even though he is being given only a weak mixture of nitrous oxide he falls into what appears to be a natural sleep and he describes it as such when he wakes up. If a nervous patient dozes off for a short period he may be alarmed when the analgesic state is entered again unless he is reassured.

The after effects of analgesia in dentistry are negligible if it is not continued longer than about ten minutes. Prolongation of this time may be followed by vomiting particularly if the posterior pharyngeal wall is irritated by dust particles or the sponge. There may be no warning signs. The patient appears to be sleeping comfortably when suddenly one or two contractions of the diaphragm occur and are followed by vomiting which is projectile in type. The dentist should not succumb to the temptation to prolong the sitting much longer merely because analgesia has already progressed smoothly for seven or eight minutes.

### SELF-ADMINISTERED ANALGESIA

Self administration of nitrous oxide or other inhalation anæsthetics for the production of analgesia is becoming increasingly popular in midwifery in surgery to allow the toilet of wounds to be carried out painlessly and in dentistry.

Various machines have been devised to enable a co operative patient to enjoy with safety the advantages of this state even though the practitioner devotes only a small part of his time to its production and maintenance. The way in which safety is achieved varies in different machines but the main principles involved are *with nitrous oxide* (i) air inlet ports are so arranged that the patient cannot inhale nitrous oxide without at the same time drawing in air through the ports or (ii) although the patient at first breathes undiluted nitrous oxide the reservoir becomes exhausted by two or three full breaths after which air only is sucked in through an inspiratory valve and *with chloroform or trilene* (iii) the patient by squeezing a hand bulb forces air through a small head of the liquid anæsthetic and the vapour

freely diluted with air is inhaled. The strength of anæsthetic vapour inhaled is controlled in part by the vigour with which the bulb is squeezed and the supply ceases altogether if the patient falls asleep (iv) A mask applied firmly to the face has two inlet tubes, normal respiration takes place solely through the short one which is open to the air, but a deepening of respiratory effort causes anæsthetic vapour to be drawn through the second tube (fig 235)

### Minnitt's Gas and Air Apparatus

The machine is of the intermittent flow type, i.e. reducing valves are so arranged that gas is liberated only during inspiration. The valve

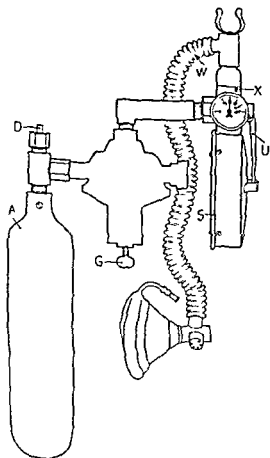


FIG 228

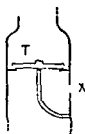


FIG 229

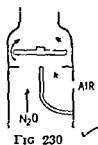


FIG 230

mechanisms are illustrated and described on pp 336 and 337. Minnitt has modified the mechanism shown in fig 225 so that when the patient breathes in gas from the reservoir bag, he must at the same time draw in air from the room. The outlet from

the  $N_2O$  reservoir bag (Q fig 225) is obstructed by a metal disc T. When the disc is on its seating (fig 229) it both obstructs the outlet of the  $N_2O$  reservoir bag and prevents the ingress of air to the patient

through five small holes (only one marked  $\lambda$  is seen in figs 229 and 230) When the patient inspires a negative pressure is created *within the breathing tube* (W, fig 228) and the weight valve T is raised G is now flows in from the reservoir bag and air is drawn in through the air ports (fig 230) The size of the air ports determines the concentration of the resulting N O/air mixture The Minnitt machine aims at delivering nitrous oxide 50 per cent air 50 per cent — a mixture which provides adequate analgesia for the great majority of patients

Minnitt's apparatus is recognised by the Central Midwives Board for use under certain conditions by midwives and of necessity it errs on the side of safety Since patients show individual variations and the machine is standard the degree of analgesia must be imperfect for the resistant subject The main criticism that can

be levelled against the apparatus is that since the mixture delivered to the patient contains 10 per cent oxygen analgesia is established relatively slowly and is therefore ineffective for a woman in whom labour pains are abrupt in onset and short lasting

#### Chassar Moir's Apparatus (fig 231)

A reducing valve is fitted to the cylinder and the gas bag reservoir fills slowly through a very fine jet which is automatically shut off when the bag is full When the patient holds the mask to her face she inspires undiluted  $N_2O$  until the bag is empty after which air is inhaled through the inspiratory valve X As soon as the bag has been emptied it begins slowly to fill By this method analgesia is quickly established but its duration is limited

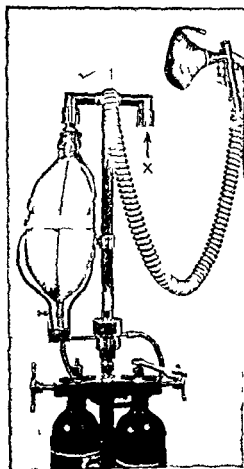


FIG 231

### Junker's Bottle

This is connected by rubber tubing to an open face mask covered with lint. The patient by squeezing the hand bulb forces air through

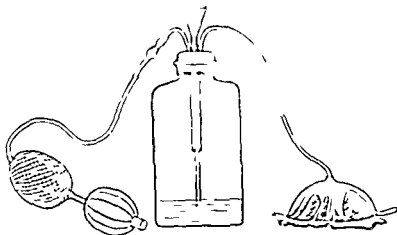


FIG 232

liquid chloroform the vapour is carried to the mask and inhaled freely diluted with air which comes through the lint. The strength of the vapour inhaled depends on the vigour of the pumping the temperature of the liquid chloroform, the amount of liquid in the bottle and on the thickness of the lint.

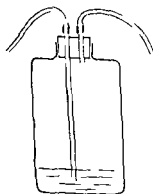


FIG 233

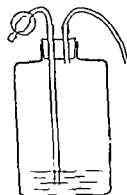


FIG 234

With a bottle of the design of fig 233 liquid chloroform instead of vapour will be blown on to the mask or patient's mouth if the tubes are wrongly connected. To prevent this accident various safety devices have been suggested and one of them is illustrated in fig 234

When the tubes are connected correctly the ball is raised from its seating when air is pumped through the bottle but if the tubes are wrongly connected it prevents the downward passage of liquid chloroform. Whatever safety device is incorporated the apparatus must be tested every time before use.

### Young Simpson's Inhaler

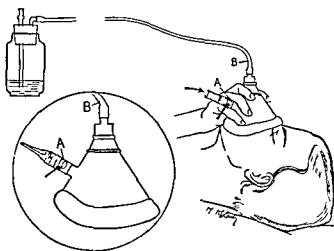


FIG 235

The short tube (A) made of celluloid is prolonged for about an inch by very thin soft rubber tubing drawn over its end. The long tube (B) is connected to the bottle containing the anæsthetic.

The patient applies the mask firmly to the face. Normal respiration takes place solely

through the short tube which has two openings to the air (see arrows fig 235). The distal opening consisting of the free rubber end, remains patent when inspiration is quiet but is closed by the approximation of its sides during a vigorous inspiratory effort (inset). Anæsthetic vapour is now drawn in through the long tube.

## CHAPTER XXXII

## THE OXFORD VAPORISER

THE Oxford Vaporiser <sup>(1 2 3 4 5)</sup> with appropriate modifications, can be used for the administration of any liquid anæsthetic \* In this

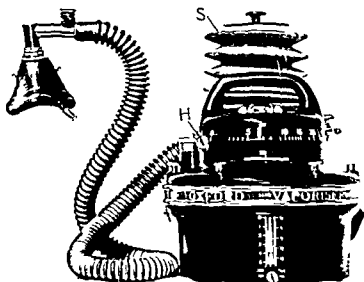


FIG 236

S hand bellows or spring bag

H control handle this corresponds to tap H in fig 240

The figures on the scale represent closely the volumes per cent of ether vapour in the ether/air mixture delivered

chapter the principles on which the vaporiser works are illustrated by showing how it is used to volatilise ether

\* If the vaporiser is to be used for chloroform a very considerable modification must be made in the temperature at which the apparatus is maintained



For reasons which will be apparent later the apparatus shown in fig 237 is not suitable for producing deep anæsthesia with ether

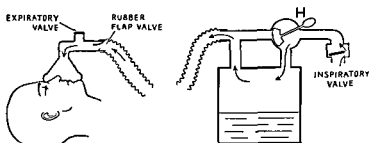


FIG 237 —A simple type of vaporiser

**Inspiration**—When the mask is held firmly to the face the inspiratory valve is raised and air is drawn in. By means of the tap H the air can be directed straight to the patient or as above diverted over the top of the liquid anæsthetic before it reaches the patient. With the tap in an intermediate position (see fig 242) part of the air can be made to travel through the anæsthetic container the remainder being by passed direct to the patient.

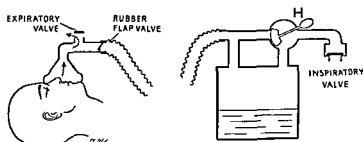


FIG 239

**Expiration**—The inspiratory valve and the rubber flap valve close; expiration takes place through the expiratory valve.

## VAPOUR PRESSURE OF LIQUID ETHER

The concentration of vapour in the saturated atmosphere above the surface of a liquid depends solely on the temperature of the liquid. If the temperature changes the concentration of vapour in the air above the liquid changes accordingly.

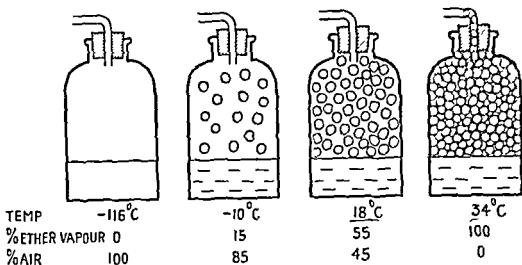


FIG 239 —To show the approximate percentage of ether vapour in the atmosphere immediately above ether at various temperatures ranging from  $-116^{\circ}\text{C}$  (freezing point of ether) to  $34^{\circ}\text{C}$  (boiling point of ether)

If the temperature of a liquid anæsthetic in a container can be kept constant there will be above it a supply of anæsthetic vapour of constant concentration

A considerable amount of heat is required to convert liquid ether into ether vapour at the same temperature. When ether is administered from a Boyle's bottle (fig 57 p 177) or from an inhaler such as that shown in fig 237 this 'latent heat of vaporisation' is not supplied from an outside source and therefore is taken from the liquid ether itself. the temperature of the ether falls accordingly and with it the percentage of ether vapour in the air immediately above it.

In the Oxford Vaporiser a supply of heat is available so that the temperature of the liquid ether and consequently the concentration of ether vapour above it is kept practically constant despite loss of heat due to evaporation. Since the concentration of ether vapour in the apparatus is high and constant the anæsthetist by directing the patient's inspired air partially or wholly over the surface of the ether, can adjust the percentage of ether vapour delivered to the patient to any desired and known level.

The use of an electrical thermostat to maintain the temperature of the ether constant was ruled out owing to the danger of explosion. Instead use has been made of the natural energy reservoir afforded by the crystallisation of a molten substance, in this case calcium chloride. When crystalline calcium chloride is heated its temperature rises uniformly until its melting point is reached when despite continued heating the temperature of the substance remains constant until it is

all transformed into liquid after which the temperature of the liquid would rise again if the heating were continued. A considerable amount of heat is taken up by the calcium chloride during its conversion from the crystalline into the liquid state *at the same temperature* and on cooling, this latent heat of fusion is given out by the substance when it crystallises (i.e. freezes) again

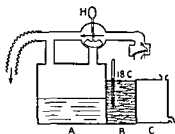


FIG 240

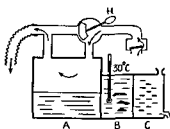


FIG 241

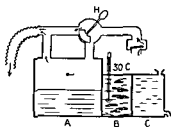


FIG 242

At the outset at room temperature say 18° C all the calcium chloride is in a solid state (fig 240) Sufficient hot water is poured into compartment C to raise the temperature of the calcium chloride in the middle compartment to melting point 30° C and then to melt a large part of the calcium chloride (fig 241) In this way the temperature of the adjoining ether container is raised to approximately 30° C

As the patient draws air through the chamber A causing ether to evaporate the fall in temperature which would otherwise occur is prevented because the heat lost by the liquid ether is immediately restored from the molten calcium chloride a fraction of which goes back into the solid state. That part of the calcium chloride which crystallises gives out its latent heat of fusion (or melting) and this is absorbed by ether in compartment A. In this way the temperature of the liquid ether and with it the concentration of the ether vapour/air mixture in the vaporiser is constant as long as some of the calcium chloride remains in a liquid state

The partially molten calcium chloride in the intervening compartment acts as a heat buffer, taking up heat from the water at any temperature above  $30^{\circ}\text{C}$  but releasing heat to its surroundings so that their temperature is maintained effectively constant at  $30^{\circ}\text{C}$

### USE OF THE HAND-BELLOWS

A spring bag reservoir or hand bellows (S figs 236 and 243) is incorporated between the vaporising chamber and the patient. When the bellows expands it draws in air through the inspiratory valve

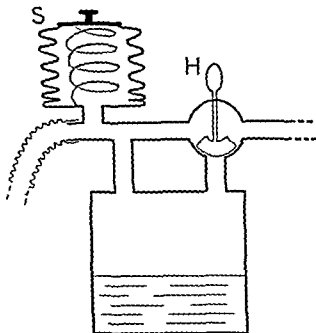


FIG 243

(fig 237) and what part, if any, of this air is directed through the vaporising chamber is determined by the position of the tap H. The bellows is not an essential part of the apparatus, but it is of value in certain situations. *It is in no way a rebreathing bag* since a one way rubber flap valve situated near the mask ensures that expirations are directed entirely through the expiratory valve (fig 238)

(i) The fact that the patient is breathing can be appreciated at a distance by watching the movement of the bag. On inspiration the bag empties slightly but the recoil of the spring within it causes it to re expand, drawing air through the inspiratory valve

(ii) In case of necessity the bellows can be used to perform

all transformed into liquid, after which the temperature of the liquid would rise again if the heating were continued. A considerable amount of heat is taken up by the calcium chloride during its conversion from the crystalline into the liquid state at the same temperature and on cooling, this latent heat of fusion is given out by the substance when it crystallises (i.e. freezes) again

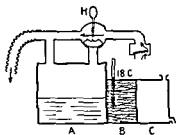


FIG 240

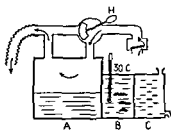


FIG 241

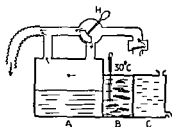


FIG 242

At the outset at room temperature say 18° C all the calcium chloride is in a solid state (fig 240). Sufficient hot water is poured into compartment C to raise the temperature of the calcium chloride in the middle compartment to melting point 30° C and then to melt a large part of the calcium chloride (fig 241). In this way the temperature of the adjoining ether container is raised to approximately 30° C.

As the patient draws air through the chamber A causing ether to evaporate the fall in temperature which would otherwise occur is prevented because the heat lost by the liquid ether is immediately restored from the molten calcium chloride a fraction of which goes back into the solid state. That part of the calcium chloride which crystallises gives out its latent heat of fusion (or melting) and this is absorbed by ether in compartment A. In this way the temperature of the liquid ether and with it the concentration of the ether vapour/air mixture in the vaporiser is constant as long as some of the calcium chloride remains in a liquid state.

(v) In certain cases to hasten the onset of surgical anaesthesia Where induction has been carried out by a moderately large dose of a short acting intravenous barbiturate respiration is depressed The patient's larynx can be accustomed to ether vapour, or the lungs may be inflated with the anaesthetic mixture by setting the control handle to the appropriate position and working the hand-bellows By the time normal respiration is resumed, a high percentage of ether vapour will be tolerated by the larynx

## THE OXFORD VAPORISER USED WITH ETHER

No matter how it is administered ether vapour is irritant to the larynx If a concentration greater than can be inhaled comfortably is administered the laryngeal defence mechanism will be set in action, with resulting coughing or laryngeal spasm The art of ether anaesthesia therefore is largely that of subduing a patient with a non-irritant anaesthetic until anaesthesia is deep enough for him not to resist inhaling a concentration of ether vapour sufficient to provide the required depth of surgical anaesthesia

On the rare occasions when ether is used for induction as well as maintenance of anaesthesia the anaesthetist should proceed as in paragraph III on p 352

Frequently unconsciousness is induced with thiopentone bromethol or nitrous oxide and the onset of surgical anaesthesia is facilitated by introducing the induction bag (1 b fig 245) between the face piece and the expiratory valve The face piece is in circuit with the ether vaporiser throughout but the induction bag can be cut off from the circuit by moving the lever to the OFF position When the lever is in the ON position the induction bag is fully in circuit with the face-piece so that rebreathing takes place freely into the bag The amount of rebreathing which takes place in and out of the induction bag is determined by the position of the lever

Ethyl chloride can be sprayed through the opening in the cap (fig 246) or the cap can be removed and vinylene or other liquid anaesthetic poured in (fig 247)

The lever of the induction bag is gradually moved into the ON position and in this way the patient comes under the influence of whatever anaesthetic is in the induction bag As soon as anaesthesia deepens sufficiently the control handle of the vaporiser (H figs 236 and 240) is set at the appropriate position commonly 10-15 and the lever of the induction bag gradually moved to the OFF position If the concentration of ether vapour is too high to be tolerated comfortably

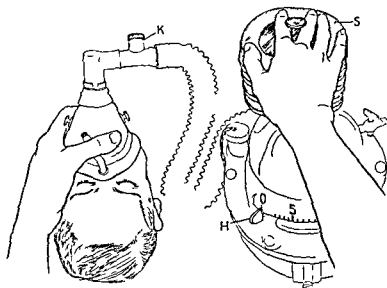


FIG 244

artificial respiration The tap H is set so that air only is drawn into the bag The expiratory valve K is screwed down and the mask is held firmly to the face with the jaw drawn forward so that the airway is clear On pressing down the bellows air will be forced into the patient's lungs The mask should now be lifted momentarily to allow expiration due to the elastic recoil of the lungs to take place This manoeuvre is repeated as often as is necessary

(iii) When anæsthesia is to be induced with ether only The mask is placed lightly on the patient's face The tap H is set so that only a small part of the air drawn in passes over the surface of the ether and the resulting weak mixture of ether vapour in air is delivered to the patient by the anæsthetist compressing the bellows When the bellows is released the spring causes it to re expand drawing in a fresh supply of ether/air mixture The tap (or control handle) is moved so that the concentration of ether vapour in the mixture is increased steadily and as rapidly as the patient will tolerate it As soon as the patient is unconscious the mask is applied firmly to the face it is now no longer necessary for the anæsthetist to deliver the anæsthetic mixture to the patient by compressing the hand bellows since the patient will draw in the mixture himself

(iv) The bellows can be used as a *vis a tergo* to deliver the ether/air mixture to the patient when the mask cannot be maintained on the face as for example when a Davis gag is used during tonsillectomy

## REFERENCES

- 1) Macintosh R R and Mendelssohn K 1941 *Lancet* 2 61
- 2) Epstein H G Macintosh R R and Mendelssohn K *Ibid* 62
- 3) Cowan S L Scott R D and Suffolk S F *Ibid* 64
- 4) Epstein H G and Pask E A *Ibid* 66
- 5) *The Oxford Vaporiser* Issued by the Nuffield Department of Anaesthetics University of Oxford



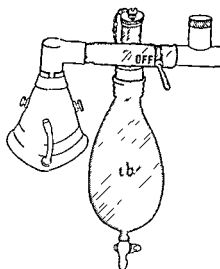


FIG 245

the induction bag can again be brought into circuit for as long as is necessary

When the patient inhales freely the desired strength of ether vapour the induction bag is removed and the depth of anæsthesia is regulated by the position of the control handle

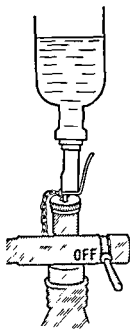


FIG 246

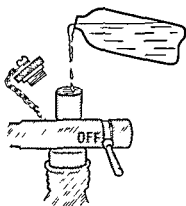


FIG 247

## REFERENCES

- (1) Macintosh R R and Mendelssohn K 1941 *Lancet* 2 61
- (2) Epstein H G Macintosh R R and Mendelssohn K *Ibid* 62
- (3) Cowan S L Scott R D and Suffolk S F *Ibid* 64
- (4) Epstein H G and Pask E A *Ibid* 66
- (5) *The Oxford Vaporiser* Issued by the Nuffield Department of Anaesthetics University of Oxford

## CHAPTER XXXIII

## LEGAL

THE law, in laying down but few principles regarding the responsibility of the anæsthetist for accidents, both grave and trifling allows the judge and jury considerable latitude in the conduct of any particular case. Legally it is not even necessary for the administrator of an anæsthetic to possess a medical or dental qualification though Halsbury states "A man should not however, undertake to do work of skill unless he is fitted for it and it is his duty to know whether he is so skilled or not" <sup>1</sup>

Although a doctor or dentist is presumed to have had the training necessary to enable him to anæsthetise for dental operations with fair *reasonable and competent skill* mere possession of a registrable qualification is not in itself either proof of ability or sufficient answer to a charge of incompetence or negligence. The law grants certain privileges to the medical and dental professions and exacts certain standards of practice. The practitioner's unwritten contract with the patient is that the former will exercise reasonable care and average skill. A practitioner who has not given anæsthetics for many years may not possess the expected degree of skill and is advised to decline to administer an anæsthetic except in case of emergency. If the patient pays for a specialist in anæsthesia the standard of skill which he is entitled to expect is as high as the latter professes to possess. The fact that a fee is not charged in no way diminishes the anæsthetist's responsibility to the patient. This applies equally to private or hospital practice.

*Production and supervision of anæsthesia is a serious undertaking*. Although a registered dental or medical practitioner is within the law if he anæsthetises his patient as well as operates on him he should act in this dual capacity only in case of absolute necessity and never in order merely to earn an additional fee nor when the services of a competent anæsthetist are available and the patient can pay for them. If a claim against the dentist arose out of an accident occurring under such conditions he might have difficulty in establishing that he was justified in fulfilling the functions both of operator and anæsthetist. There are indeed circumstances which justify the dentist in administering the anæsthetic to his own patient. Thus the financial position of

the patient may make this course necessary, or the only practicable alternative may be to call in an inadequately skilled family doctor, who might prove more of a hindrance and even a danger than a help. Blame for failure to extract the tooth successfully is almost invariably attributed to the extractor and not to the anaesthetist! The family doctor who in his student days may never have seen a skilled dental anaesthetist at work, often fails to realise his own incompetence in this field. If he anaesthetises the patient too lightly or too deeply he embarrasses rather than helps the extractor. The dentist who gives his own anaesthetic has generally had the advantage of a proper training in this specialised branch of anaesthesia in his student days and he may even have taken post-graduate instruction in the subject. After he has induced smooth anaesthesia he is able with the assistance of a nurse or mechanic to steady the head to operate more efficiently and with greater peace of mind than if the patient were in the charge of a medical practitioner less experienced in dental anaesthesia than the dentist himself.

Provided the dentist has been trained in the administration of anaesthetics there is no reason why he should not act as anaesthetist for extractions by a colleague but he should confine himself strictly to the use of drugs with the action of which he is familiar. Normally these would be nitrous oxide and ethyl chloride. Ether is generally considered to be outside the dentist's province. In our view, chloroform should never be used for dental operations. We also think it inexpedient for a dentist to administer any intravenous anaesthetic although if he has familiarised himself with the associated dangers and difficulties there is no logical reason why he should not do so.

When the dentist's nurse or mechanic assists in giving the anaesthetic responsibility for the choice of the anaesthetic and its administration lies entirely on the dentist but if the operator has the help of a qualified dentist or doctor as anaesthetist the responsibility devolves upon the latter whose liability in this respect does not differ from that assumed in administering any other drug. Since it may be necessary to modify the anaesthesia at a moment's notice the anaesthetist must always be free to act as he thinks advisable in the circumstances. The onus both for the choice of anaesthetic and for any damage directly attributable to its use must be borne by the anaesthetist and cannot in ordinary circumstances be made to devolve on other parties. The anaesthetist should therefore not employ any anaesthetic procedure of which he does not himself entirely approve

Legal actions where the claim is made that the patient has suffered

because due care and skill have not been exercised by the anæsthetist and dentist are not infrequently successful. One of the most serious accidents that can befall a patient during a dental operation is the entry of a foreign body into the air passages. This may occur without dentist or anæsthetist becoming aware of the accident and it is not necessarily the fault of either. Although in fact neither may be morally or actionably blameworthy there is a strong presumption that one or other of them is culpable and if the accident occurs in the absence of reasonable precautions there may be no defence at law. Even if proper precautions have been taken the dentist is in the wrong if after an operation and before removing the pack he fails to make sure no foreign body is in the mouth and if he fails to examine the teeth remaining in the mouth and those extracted to see if any tooth or portion of a tooth is not accounted for. Further when the pack is removed the operator should again satisfy himself that no loose body remains in the mouth or oro pharynx. Finally the dentist is blameworthy if he fails to take proper action if any tooth or portion of a tooth is found to be or suspected to be missing.

During a dental operation the three faults which most frequently contribute to the entry of a foreign body into the lungs are failure to insert a mouth pack in such a position that the laryngeal opening is reasonably guarded, operating on a patient when he is restless and failure to remove immediately from the mouth any dislodged tooth, tooth fragment or filling. Packing off the mouth is of paramount importance and it is a universally accepted rule that no operation in the mouth under general anæsthesia should be undertaken until the larynx has been protected as adequately as possible by a pack against the entry of a foreign body. The efficiency of the pack varies in different circumstances.

For short operations in the dental chair it is quite impossible to arrange the mouth pack so as to provide absolute protection. For this ideal to be achieved the larynx would have to be occluded and the airway thus obstructed. If the pack is placed too far back retching reflexes may be initiated and make the operation practically impossible. At the same time the soft palate may be pushed against the posterior pharyngeal wall and seriously impede nasal respiration. For simple extractions a coarse meshed marine sponge provides a very high degree of protection as long as the dentist ensures that it is placed advantageously throughout the operation and that it is replaced by a fresh sponge as soon as it becomes soaked in blood and mucus which much reduces its protective properties. The pack is sometimes provided by the anæsthetist and often inserted by him into the

posterior part of the mouth before the operation begins. The anæsthetist almost invariably stands behind the patient and therefore cannot follow the changing conditions in the mouth so that the dentist, as soon as he begins to operate, assumes responsibility for the position of the pack even when he did not insert it himself. He often finds it necessary to change its position to meet the needs of the operation, and he must bear the responsibility for the consequence of any such change.

When an anæsthetic is given badly the patient may be restless or struggle, thus increasing the chances of a foreign body slipping out of the forceps, an accident which can occur even in the most favourable circumstances. Then, unless the throat has been adequately packed, there is a danger of the loose body entering the trachea. If the responsibility here must be borne entirely by one person, we regard it still as belonging to the dentist, for he should decline to operate if the anæsthesia is inadequate.

Major dental operations present totally different problems. These are generally performed at home or in hospital, often under ether anæsthesia. The deeper the unconsciousness, the more nearly complete is the abolition of all reflexes, including the protective cough reflex, and therefore the more essential it is to safeguard the larynx. The only sure way of securing the patient's safety is to administer the anæsthetic through an endotracheal tube and then to pack the throat in such a way that the larynx is completely protected. Unless this procedure is adopted, an extensive jaw operation in the supine position can easily degenerate into a shambles. Conditions are equally unsatisfactory for both dentist and anæsthetist. The anæsthetist has difficulty in maintaining a clear airway and may be unable to provide smooth anæsthesia. If even a small degree of respiratory obstruction occurs, it produces venous congestion and increases hæmorrhage, and therefore the dentist often has to work by sense of touch in a pool of blood. When the field of operation is thus obscured by hæmorrhage, the danger of entry of a foreign body is always much increased. This is especially so if the patient is in the supine position. Periodically the operation should be suspended and blood and debris cleared away, but even when this precaution is taken, the situation is potentially dangerous, and it may be further complicated by vomiting. Anyone who has witnessed a major dental operation or any other prolonged operation on the jaw in these circumstances will realise that the stage is well set for an accident. In an action arising out of the lodgment of a foreign body in the lungs in these circumstances, if a jury exonerate the anæsthetist from blame for not providing endotracheal anæsthesia,

they might as well toss a coin in deciding how to apportion blame between anæsthetist and dentist

Greater anæsthetic skill and experience are required for major dental operations than for the average operation for say herniotomy or appendicectomy Multiple extractions are seldom emergency operations and we feel that the doctor should avoid anæsthetising for such cases if he cannot pass an endotracheal tube He would not undertake any other medical procedure unless he could carry it out proficiently neither should he anæsthetise in circumstances where he cannot provide both safety for the patient and the operating conditions which a dentist is entitled to expect We realise that endotracheal anæsthesia is at present regarded as being beyond the province of a general practitioner but as medical knowledge grows the boundaries of legal negligence expand We believe that in the future it will be held to be negligent to perform a major jaw operation without endotracheal anæsthesia

In an operation under endotracheal anæsthesia the anæsthetist can pack off the back of the throat in such a way that it is impossible for a tooth tooth fragment blood clot or any other foreign body to enter the larynx and it is customary for him to undertake this duty Before beginning to operate the dentist should verify that such protection is complete and thereafter assume responsibility for the position of the pack

It is a counsel of perfection that the portions of a tooth broken during extraction should be pieced together by the dentist and a search made for any missing fragments We should record that in a very large number of cases at which we have assisted the tooth and filling have broken into so many pieces that an attempt to follow this advice would have been ludicrous In the difficult question of the correct procedure after such an event the dentist's own conscience is his best guide as to what course to take If the dentist considers that the pack has been effectively placed and if he cleared away debris at once when the tooth fragmented usually he feels satisfied that there cannot be a foreign body inside the patient and if he has this conviction no further action need be taken When the dentist has reasonable grounds for suspecting that a foreign body is lodged somewhere inside the patient he should adopt a line of action which will ensure the patient's safety The policy of trusting to luck and of saying nothing is in these circumstances entirely reprehensible for it may lead to the death of the patient The question of whether extensive radiological examination of the patient's head chest and abdomen should be undertaken must first be decided Some teachers hold that this should be done in every case

where a tooth fragment remains unaccounted for. It is our opinion that an X ray examination should be made if the missing fragment is considered large enough or characteristic enough to throw a *recognisable* shadow but that if it is thought unlikely that the fragment would be recognisable radiologically such an examination is not necessary, indeed it may even prove misleading since shadows at the hilum of the lung may rouse the suspicion that a foreign body is in the thorax when this in fact is not the case. Also unless the missing object is metallic negative radiological findings are not conclusive evidence that there is no foreign body inside the patient. We have known radiological examination to be ordered by the dentist when only a small fragment of tooth was missing solely to protect himself in the event of subsequent litigation, and this in the case of a patient suspected of an inclination for redress may well be a desirable precaution since by some it is regarded as supporting evidence that the dentist has made every effort to safeguard his patient.

The treatment of a misplaced fragment, the location of which is identified after the accident is discussed on p 309. If radiological examination is considered unnecessary or if it is undertaken and fails to reveal the situation of the foreign body the patient or a sensible relative and also the family doctor should be informed of what has happened. Bulky residue foods should be advised for about a week in case the foreign body has been swallowed (p 307). Any chest complaint which does not clear up must be regarded with suspicion since it may be the beginning of a lung abscess. If an action should arise for damages due to a foreign body in the lung and if the dentist has not taken the course indicated above he will find himself with no defence because he has not acted with the reasonable judgment and care to be expected from him. The following instances in our own experience illustrate these points.

1 On many occasions when one or other of us gave the anaesthetic a small piece of tooth has not been accounted for. On each of these occasions it was thought that the missing fragment was too small to be recognisable radiologically among the shadows cast by ribs and by glands in the hilum of the lung. It was considered correct not to X ray the patient nor to acquaint him with the full implications of the situation as this knowledge would be an unnecessary mental burden to a nervous patient. He was told to report at once if he developed a cough during the next few months and at the same time full details were communicated to the family doctor. In none of these cases did a pulmonary lesion subsequently develop so it can be assumed that



the missing fragments were either swallowed or were lost outside the body

2 The writers so far as they know have anæsthetised only one case in which a foreign body has entered the lung. An upper eight was being extracted under nitrous oxide in the dental chair. As the extraction proved difficult the anæsthetist lowered the head rest and extended the head to provide better access. After some minutes the tooth was extracted and dentist and anæsthetist congratulated each other on a job well done. Anæsthesia was tranquil throughout and the patient did not cough at any stage. The man continued at work and four weeks later he became ill and influenza was diagnosed. The pulmonary condition did not improve and an X ray examination disclosed a foreign body located in the lungs. This was identified as a piece of amalgam filling which had been dislodged unnoticed from the adjoining molar tooth during the course of extraction. Arrangements were made for bronchoscopy to be performed. On the previous evening however the patient rolled over in bed and the sudden change in position presumably moved the foreign body. This precipitated a bout of coughing during which the filling was expelled with such force that it excoriated his soft palate. A claim for damages was made and as the dentist had not examined the mouth after the extraction the patient was deemed to be justified in his claim and the matter settled out of court.

3 Only once have we thought it necessary to have the patient X rayed directly after the operation. A lower wisdom tooth broke while it was being extracted. An elevator was then used after which the root socket was found to be empty but the root could not be found. X ray examination revealed that it had slipped under the mucous membrane distal to the socket. It was removed with ease without further anæsthesia.

An anæsthetist may sometimes be held responsible for damaging existing teeth or bridge work. Before beginning to induce anæsthesia he should therefore examine the patient's mouth and make a point of noticing any loose teeth, crowns or bridge work. He should be particularly careful to avoid using a Mason's gag on such structures. He may also damage the front teeth when he uses a laryngoscope in order to pass an endotracheal tube. The risk of this accident is diminished by providing anæsthesia of sufficient depth to relax the jaw and by protecting the front teeth with a thin sheath of malleable lead or adhesive plaster.

Actions claiming damages against dentists and anæsthetists for fracture of the jaw occurring during the course of extraction are not

infrequent That such accidents can occur without carelessness on the part of either makes it all the more imperative that a Mason's gag should be used with great care particularly when the jaw is known to be fragile

One of us has anaesthetised five cases in which the jaw was broken during dental extractions Although in four no undue force was used, nevertheless the jaw broke during the actual extraction In the fifth a large amount of bone covering an unerupted lower wisdom tooth on the right side was removed from a powerful man This tooth was extracted successfully, and it was decided to proceed with a similar operation on the opposite side During the use of hammer and chisel on the left side the jaw fractured on the right side where it had been weakened by the extensive removal of bone A Mason's gag was not employed in any of these cases, but if it had been used and used roughly it is easy to understand how it might have caused or contributed to this accident

In inquests on deaths during anaesthesia the anaesthetist is always asked if he examined the patient and if he can state that he applied a stethoscope to the heart and chest he is considered to have done his duty although such brief examination as is possible in the dental surgery is, in fact valueless If it could be expected to assist in any way we would be very much in favour of it and we think it worth recording that it is a common custom for the anaesthetist to go through the formality of examining the patient simply to protect himself at law For this purpose we have known the mere pretence at examination to be made, the stethoscope being applied outside the coat and the patient then pronounced fit We believe that a patient who does not volunteer a history of illness and who is able to walk into the dental surgery can be presumed fit enough to submit to a short dental anaesthetic The anaesthetist should be able to form an opinion of the patient's general condition at a glance but in any case the history yields much more valuable information than does any cursory physical examination The patient may for example reveal the presence of myocardial degeneration by saying that he cannot walk upstairs without becoming short of breath whereas an ordinary routine examination of the heart may disclose nothing We do not believe that any routine examination such as would normally be made by an anaesthetist in the dental surgery is in any way helpful and therefore we do not consider it to be negligence to omit examining such a patient before an ordinary short dental extraction

One disadvantage of a routine examination is that some patients do not realise that it is routine and are alarmed fearing that it indicates misgivings about their health. Others interpret a physical examination to mean that they are going to be exposed to a greater strain than is in fact the case. On the other hand there are patients who welcome a brief examination and even ask for it in which case it should not be omitted. There are some anæsthetists who believe that every patient about to be anæsthetised is worried about his heart and that it is therefore desirable to apply a stethoscope if only to create an opportunity for reassuring the patient. The only substantial reason for the examination of an apparently fit patient before a short anæsthetic in the dental surgery is that it will contribute to his peace of mind and thus facilitate anæsthesia. If it is considered that it will not achieve this object it should be dispensed with. In a case where the patient is examined he should be reassured as to his general fitness before the anæsthetic is begun.

The following warnings embody the ideals which should be aimed at in every case though it is recognised that in certain instances they may not be easy of attainment. By attending to these points as carefully as he can the anæsthetist will do much to protect himself against litigation.

**Never if it can possibly be avoided act both as dentist and anæsthetist**—Administration of an anæsthetic is a full time job.

**Never administer an anæsthetic unless a third person is present**—Observance of this rule safeguards the administrator against false charges of misconduct. The presence of a third person is an additional advantage inasmuch as he can be called upon to assist in emergency.

**Never anæsthetise a patient unless his consent to be anæsthetised has been given**—When the patient is under 21 he is legally a minor and the permission of the parent or guardian should be obtained.

From the legal point of view the dentist and anæsthetist should have prior authority in writing, for performance of the operation and administration of the anæsthetic. In private practice it is most unusual for this written permission to be asked for and the patient's assent to be anæsthetised is therefore commonly taken as being implied.

**Never give an anæsthetic unless adequate first-aid equipment is to hand**—A Mason's gag and tongue forceps are essential.

**Never leave an unconscious patient without a trained person to look after him**—It is the anæsthetist's duty to see that the patient is safeguarded until his return to consciousness. If recovery

is likely to be protracted it is customary for this responsibility to be delegated to a nurse. The exigencies of a busy operating day in hospital seldom permit the anaesthetist to accompany the unconscious patient from the operating theatre to his bed although in private practice he should always do so. The anaesthetist should then satisfy himself that the patient is protected from being burnt by hot-water bottles that his position is such that his airway will not become obstructed and that any blood, mucus or vomitus will run out of the mouth instead of being aspirated. A Mason's gag and tongue forceps should be kept by the bedside until the patient is conscious. Finally, before the anaesthetist leaves he should make it clear to the nurse that the responsibility for the patient is being left in her hands (p 286).

One of the first expenses which the young medical or dental practitioner should incur is to join one or other of the recognised defence organisations viz the London and Counties Medical Protection Society, Victory House Leicester Square London WC 2 the Medical Defence Union Ltd 49 Bedford Square London WC 1 or the Medical and Dental Defence Union of Scotland Ltd. The public is becoming increasingly litigation minded and if everything associated with an operation does not proceed exactly as hoped for a patient is sometimes encouraged to believe that he is entitled to damages. In such circumstances the patient has much to gain and little to lose but the practitioner is in exactly the reverse position. In order to prevent his name figuring in the Law Courts he may be blackmailed into paying large damages even where none have been incurred either legally or morally. Any of the defence organisations is prepared to defend with the utmost vigour the practitioner who finds himself in this situation and if necessary is in a position to take a case to the House of Lords. Unjust claims have often been withdrawn as soon as it became known that the practitioner was protected by a powerful Society and in defending claims which have some appearance of being justifiable the practitioner has at his disposal the vast experience of the Society.

#### REFERENCE

- (1) Halsbury 1912 *The Laws of England* 21, 369

## INDEX OF PERSONAL NAMES

*The figures in italics refer to pages where the personal name occurs in References*

- Ackland W R 271 272  
 Adam 337  
 Archer W H 297 304  
 Auer J 10
- Baetzner W 11 12  
 Bannister F B 164 269 270  
 Bayliss L E 53  
 Beddoes T 2 11  
 Bell I R 169  
 Benedict F G 89  
 Berger I M 203  
 Bernard C 188 202  
 Blomfield J 12  
 Bostock J 11  
 Boston F K 169  
 Breuer J 50  
 Bradley W 203  
 Brunton T L 280  
 Buller A J 196 202  
 Burkhardt L 12  
 Burns B D 189 202  
 Burstein C L 151
- Chandy J 203  
 Chen M Y 169  
 Clement F W 210 245  
 Clendon D R T 203  
 Clover J T 8 12  
 Cocking T T 53  
 Cohen H 133  
 Cole F 191 202  
 Colton G Q 4 5 7  
 Constantin J D 106  
 Courville C B 123  
 Cowan S L 39 35  
 Cullen S C 202
- Dalton J 32  
 Dana R H 11  
 Davison M H A 203  
 Davy H 2 3 11  
 de Pass 278  
 Dioscorides 1 11  
 Doyen E 9  
 Drinker C K 53 333
- Du Bois E 89  
 Duisberg H 10  
 Dunham 7  
 Dutcher J D 187 202
- Edwards P M 201 203  
 Eichholtz F 10 12  
 Elsberg C A 10 12  
 Epstein H G 355
- Faraday M 6 11  
 Feldberg W 186 203  
 Forné 10  
 Freedman A 170 172  
 Freund 184
- Gelfan S 169  
 Gill R C 186 202  
 Gillespie N A 248 270  
 Goldman V 166 168  
 Gray H 149 151  
 Gray T C 196 202  
 Greenwood M 133 133  
 Griffith H R 11 12 186 188 202  
 Grove 152  
 Guedel A E 63 64 66 67 69 74  
 88 89 106 268  
 Gunther R T 11  
 Guthrie S 180  
 Gwathmey J T 10 12 19 101
- Halsbury 1st Earl 356 365  
 Halton J 188 202  
 Hayden G G 6  
 Henderson V C 184 18,  
 Henderson Y 53 206  
 Herdman K N 172  
 Hering E 50  
 Hewer A J H 200 203  
 Hewer C L 170 172 333  
 Hewitt F W 112 277 284  
 Heyfelder F 164  
 Hickman H H 3 4 11  
 Hill B 170 172  
 Hobson J A 202 203  
 Homer 1 11

Hort A 11  
Human J U 64 67  
Humphrey J H 172  
Hunter A R 203

Jackson Chevalier 10 247 248 270  
291 299 300 301 304 306 309 333  
Jackson C L 210 304 333  
Jackson C T 6  
James N R 333 333  
Johnson G E 12 202  
Junker F 12 345

Kenny W R 53  
King H 186 187 202  
Kirstein A 9 12  
Knight T A 4  
Kuhn F 9 12

Laborde J B V 313 333  
La Motte F L 53  
Langston G T 203  
Lannelongue 12  
Lavoisier A L 2  
Leake C D 165 169  
Levy A G 105 106  
Liebig J 180  
Lister J 1st Baron 183 183  
Long C W 6 11  
Lucas B G B 200 203  
Lucas G H W 184 185

Macbeth R G 269 270  
McClelland M 172  
McCrae T 333  
MacEwen W 9 12  
Macintosh R R 194 202 213 215  
245 268 270 333 355  
McKesson E I 213 315 327 336  
Macklin A H 21 28  
Macleod J J R 210  
Magendie F 2 11  
Magill I 10 12 255 312  
Mallinson F B 201 203  
Marin M G 97  
Marrett H R 171 172  
Mason D F J 202  
Mason F 210 271 284  
Meltzer S J 10 12  
Mendelssohn K 355  
Miller W S 33 53  
Minnitt R J 336 343  
Moir J C 344  
Morton W T G 5 6  
Mushin W W 202 278

Negus V E 270  
Nosworthy M D 23  
Nuffield 1st Viscount 11

Organe C S W 192 193 202, 203

Paltauf A 130 133  
Paré A 11  
Pask E A 270 355  
Paterson H J 219 245  
Paton W D M 189 202 203  
Penfold J B 203  
Peters J P 53  
Pliny the Elder 1 11  
Pratt C L G 333  
Pratt F B 245  
Pravaz C G 2  
Priscott F 191 192 193 200 202  
Priestley J 2  
Primrose W 12

Reed A B 53  
Richardson B W 8  
Riggs J M 5  
Riley H T 11  
Robbins B H 185  
Rowenstone E A 151 184 270  
Rowbotham E S 10 12 101 106  
192 193 200 202 203 255

Salt R 169  
Schaefer E A S 329  
Schimmelbusch C 158 175 182  
Schmidt E R 185  
Scott R D 164 355  
Serturmer F W A 2 11  
Shaw G B 88 106  
Shimer W 89  
Shipway F E 12  
Silverster H R 331  
Simpson J Y 7 12  
Simpson T Y 346  
Sims J M 7 11  
Sington H 160  
Snow J 8 12  
Soubeiran E 180  
Stephen C R 203  
Suffolk S F 355

Taylor F L 11  
Theophrastus of Eresos 1 11  
Thomas G J 252 270  
Thompson R C 11  
Torrens J A 201 203  
Trendelenburg F 9 195 290

Trewby J F 215 216 227 277  
 Turnbull H M 133  
  
 Van Slyke D D 53  
  
 Warren J C 5  
 Waterfall J M 203  
 Waters R M 11 176 179 184 185  
 Waterton C 186 202  
 Watt J 2  
 Wedel G W 11  
 Weese H 11 12  
 Wells C J 11  
 Wells H 4 5

West R 191 202  
 Wien R 202  
 Willstatter R 10  
 Wintersteiner 187 202  
 Winton F R 53  
 Wood A 2  
 Wood M W W 201 203  
 Woods H M 133  
 Wright 186  
  
 Young I M 196 202  
 Young M 133  
  
 Zarnis E J 202 203

## INDEX

## A

- Abdominal cavity operations in depth of anaesthesia necessary for 67  
 — operation pre anaesthetic preparation of patient for 136  
 Accidents during anaesthesia 356-363  
 — — — prevention of 363  
 Acetylcholine 189-191  
 Ackland's blades 271-272  
 Adam's reducing valve 337-338  
 Adrenaline in treatment of cardiac failure 333  
 Air administration of during nitrous oxide anaesthesia 231  
 — alveolar 33  
 — — composition of 33  
 — — oxygen tension of 36  
 — dead space 31  
 — passages foreign bodies in 297 306-309  
 — residual 31  
 — respired volume of 31  
 — tidal 31  
 Airway 292  
 — obstruction of 218  
 — — prevention of 232  
 — restorer 292  
 Alcoholics anaesthesia in 97-99  
 Alimentary tract foreign bodies in 307  
 Alveolar air 33  
 — — composition of 33  
 — — oxygen tension of 36  
 — — partial pressure of gases in 33  
 Ambulatory patient pre anaesthetic medication for 93  
 — — preparation of for anaesthesia 134  
 Ammonia aromatic in treatment of post anaesthetic nausea 244  
 Amputations depth of anaesthesia necessary for 67  
 Anaemia and cyanosis 56-61  
 — and nitrous-oxide anaesthesia 73 74  
 — as cause of anoxia 51  
 — barbiturates contra indicated in 144  
 Anaemic patient anaesthesia in 56 74 121  
 Anaesthesia (*see also* under the names of the various anaesthetic agents)  
 — basal 99-103  
 — — contra indications to 303  
 — — first employment of 10  
 — — in children 124-126  
 — choice of local or general 75 78  
 — control of level of 15  
 — cyanosis during 39 54-61 73  
 — definition of 14  
 — delirium during 64-65  
 — dental preliminary approach to 204-210  
 — depth of determination of 63  
 — — — for different operations 66-67  
 — duration of factors governing 26  
 — endotracheal 246-270  
 — — history of 9-10  
 — — warnings on use of 266  
 — first stage of signs of 64  
 — fourth stage of signs of 66  
 — general choice of anaesthetic in 79-87  
 — — contra indications to 302-303  
 — — in dentistry indications for 76-77  
 — history of 1-12  
 — in children 124-129  
 — in obstetrics 8 342-346  
 — indications for local or general 75-78  
 — intravenous in dental surgery 86  
 — legal aspects of 356-363  
 — local 13  
 — — in combination with regional anaesthesia 13  
 — — in dentistry indications for 77-78  
 — methods of induction of 14  
 — morbid conditions simulating 14  
 — preparation of patient for 134-136  
 — production of by oxygen deprivation 22  
 — psychological disturbances during 222  
 — recovery from 240  
 — regional 13  
 — second stage of signs of 64  
 — signs of 62-74  
 — — modified by *d* tubocurarine chloride 67 193  
 — spinal 13  
 — stages of 63-67  
 — surgical signs of 65-66 70  
 — theory of 19-28  
 — third stage of signs of 65  
 — use of *d* tubocurarine chloride in 192-198  
 Anaesthetic choice of in general anaesthesia 79-87  
 — general choice of in dental surgery 84-87  
 Anaesthetic resistant patient 113  
 — — use of strap in 115 208  
 Anaesthetics absorption of by fats 21  
 — basal administration of by intravenous route 104  
 — — — by oral route 103  
 — — — by rectal route 101



Anæsthetics control of action of 15-16  
 — differential distribution of in the body 23  
 — difficulties of administration of due to patient 107-123  
 — dislike of cumulative 112  
 — effect of on physiological processes 21  
 — essential qualities of 15  
 — inhalation speed of action of 66  
 — mode of action of 19  
 — nasal administration of 222  
 — overdose of causing respiratory arrest 327  
 — — counter measures in 17 327  
 — resistance to 5 88-89 113-118  
 — — premedication in 90  
 — — use of thiopentone in 115 116 141 142  
 — respiratory exchange of 36-37  
 Anæsthetist and dentist co operation between 234  
 — consultation of 81  
 — legal responsibilities of 356-365  
 Analgesia 339-346  
 — in dentistry 340  
 — in obstetrics 342-346  
 — production of 341  
 — self administered 342-346  
 — signs of 64  
 Analgesic potions 1  
 Anoxia 50-52  
 — and cardiac failure 120  
 — as cause of anæsthesia 19-28  
 — causes of 51-52  
 — effect of on respiration 46-48  
 — in nitrous oxide anæsthesia 69-74 212 218 219  
 Apnoea 49 50  
 — due to *d* tubocurarine chloride 192 195  
 — during anæsthesia 229  
 Apparatus for use in dental surgery 213-218  
 Aryepiglottic folds œdema of causing respiratory obstruction 301  
 Asphyxia avoidance of in nitrous oxide anæsthesia by pre anæsthetic medication 94  
 — during administration of nitrous oxide 69 218 219 310-312  
 Asthma barbiturates contra indicated in 144  
 Asthmatics premedication of 120  
 Atropine 105  
 — as premedicant in ether anæsthesia 173  
 — use of with prostigmin 190 197  
 Autoscope 9  
 Avertin as basal anæsthetic for children 125  
 — discovery of 10  
 — fluid rectal administration of 102  
 — for anæsthesia of claustrophobic patient 122  
 — use of in phthisical patients 77

## B

Barbitone 137  
 Barbiturates 137-151 (*See also* under the names of individual drugs)  
 — action of in pre anæsthetic medication 92-93  
 — and laryngeal spasm 312  
 — contra indications to use of 143-144  
 — in anæsthetic resistant patient 116  
 — indications for in dental surgery 86  
 — oral administration of 103  
 — pharmacological action of 137  
 — physical properties of 137-138  
 — poisoning with treatment of 151  
 — rate of injection of 26  
 — rectal administration of 103  
 — use of as basal anæsthetics 11  
 Basal anæsthesia 99-105  
 — — contra indications to 303  
 — — disadvantages of 100  
 — — first employment of 10  
 — — in children 124-126  
 — — route of induction of 101-105  
 — anæsthetics intravenous administration of 104  
 — — oral administration of 103  
 — — rectal administration of 101  
 Bed ridden patient anæsthesia in for dental surgery 121  
 Blood buffering power of 43-45  
 — carbon dioxide carrying capacity of 39 43  
 — clot as cause of respiratory obstruction 314  
 — concentration of anæsthetic in during induction 22  
 — hæmoglobin content of 37  
 — nitrous oxide carrying capacity of 39  
 — oxygen-carrying capacity of 37-39  
 — pH 43  
 — transport of gases by 37  
 Blood pressure high anæsthesia in presence of 121  
 Boyle's bottle modified in semi-closed method 177 349  
 Brain cell anoxia of during induction of anæsthesia 24  
 — operations on depth of anæsthesia necessary for 67  
 Breath holding during induction of ethyl chloride anæsthesia 155 157  
 British Oxygen Co's reducing valve 337  
 Bromethol 102 (*See also* Avertin)  
 Bronchitis anæsthesia in 77 119  
 Bronchospasm from use of *d* tubocurarine chloride 191  
 Brunton's swivel prop 280  
 Buffer salts 41

## C

C 5 200  
 C 10 199-200  
 Calabash curare 187

- Carbon dioxide absorption technique 11  
17s  
— — and oxygen exchange 34-36  
— — contra indicated in treatment of  
overdose of anæsthetic 327  
— — role of in regulation of respiration  
46-50  
— — tension of in alveolar air and  
tissues 3s 36  
— — transport of by blood 39 43  
— — dissociation of 42  
Carbonic acid gas anæsthetic effects of 4  
— — anhydrase action of in respiration 36  
— — function of in respiration 45  
Cardiac failure anæsthesia in presence of  
120  
— — during anæsthesia 332  
— — — — treatment of 332-333  
— — puncture in treatment of cardiac  
failure 333  
Chassard Moir's apparatus use of to pro-  
duce analgesia 344  
Chest operations depth of anæsthesia  
necessary for 67  
Chevalier Jackson position 247  
Children anæsthesia in 124-129  
— — use of swivel prop in 280  
Chloral as basal anæsthetic 10  
Chloride shift 44  
Chloroform 180-183  
— — administration of 182  
— — — — by Oxford Vaporiser 347  
— — dangers of 181  
— — first use of as anæsthetic 7  
— — physical properties of 180  
Cholinesterase 189 190  
*Chondrodendron tomentosum* 187 188  
Circulation rate diminution of as cause  
of anoxia 51-52  
Circulatory diseases anæsthesia in pre-  
sence of 120  
Claustrophobics anæsthesia in 122  
Cleft palate anæsthesia in patients with  
123  
Cocaine addicts anæsthesia in 118  
— — use of in nasal intubation 249  
Colour of patient changes in during  
nitrous oxide anæsthesia 54-61 73  
Coramine acceleration of rate of recovery  
from thiopentone anæsthesia with 151  
Cotton wool mouth pack 232  
Curare 186-198 (See also *d* tubocurarine  
chloride)  
Curarine 187  
Curine 187  
Cyanosis 54-61  
— — during anæsthesia 54-61 73  
— — — — ætiology of 54  
— — — — hæmoglobin in 55-61  
— — nitrous-oxide anæsthesia 39 54 61  
73  
Cyclopropane 184-185  
— — administration of 18s  
Cyclopropane first employment of 11 184  
— — indications for use of 80-81  
— — physical properties of 184  
— — use of in anæsthetic resistant patient  
115  
Cylinder valves 334-337  
Cylinders 216  
— — estimation of contents of 211 212  
  
D  
Dalton's law 32  
Dead space air 31  
Death during anæsthesia 130-133 181  
304  
Debility barbiturates contra indicated  
in 144  
Decamethonium iodide 199-201  
Defence organisations 36s  
Delirium during anæsthesia 64 65  
Dental anæsthesia preparation of patient  
for 204-210  
— — surgery 212-245  
— — anæsthesia in children for 124-129  
— — analgesia in 340  
— — apparatus for use in 213-218  
— — — — — advice on use of 217  
— — choice of anæsthetic in 75-78 84-87  
— — indications for local or general  
anæsthesia in 75-78  
— — thiopentone in 142  
— — pre anæsthetic medication for 93-  
9s  
— — — — preparation of patient for 134-  
135  
— — — — use of vinesthene in 165-169  
Dentist and anæsthetist co operation  
between 234  
— — as anæsthetist 3s6-3s7  
de Pass prop 27s  
Diaphragm action of in respiration 29-30  
Dichloroacetylene production of from  
trilene and soda lime 171  
Diet prior to anæsthesia 134-136  
Discomfort during induction of anæ-  
sthesia prevention of 219  
Di vinyl ether 165-169  
— — administration of by closed method  
166  
— — — — by open method 168  
— — — — dosage of 166 168  
— — — — in dental surgery 165  
— — — — indications for in children 12s  
— — — — — in dental surgery 87  
— — — — physical properties of 16s  
— — — — signs of anæsthesia with 166  
— — — — use of in anæsthetic resistant  
patient 117  
Dreams during recovery from anæsthesia  
241-243  
Drug addicts anæsthetisation of 118  
*d* tubocurarine chloride 186-198  
— — — — administration of 194  
— — — — antidote to 190 197

- d* tubocurarine chloride apnoea due to use of 192 195
- — — bronchospasm resulting from 191
- — — contra indications to use of 198
- — — dosage of 193
- — — effect of an electrocardiogram 192
- — — in Caesarean section 196
- — — in children 193 194
- — — in electrical convulsion therapy 197
- — — in myasthenia gravis 198
- — — in treatment of laryngeal spasm 196 312 313
- — — in treatment of tetanus 196
- — — indications for 195 196
- — — pharmacology and physiology of 186-192
- — — signs of anaesthesia during use of 67 193
- — — use of in anaesthesia 192-198

## E

- Edentulous patient use of gag in 275
- Electrical convulsion therapy modified by use of *d* tubocurarine chloride 197
- — — — — decamethonium iodide 200
- Emergencies anaesthetic 305-333
- Emphysema as cause of anoxia 51
- Endotracheal anaesthesia 246-270
- — — history of 9-10
- — — warnings on use of 266
- — — tube use during dental operations on children 126
- Endurance reducing valve 337
- Epileptics anaesthesia in 122
- Ether 173-179
- — — administration of by closed circuit method 178-179
- — — — — by open drop method 175-176
- — — — — by Oxford Vaporiser 347-355
- — — — — by semi-closed method 177
- — — anaesthetic properties of discovery of 6
- — — contra indications to in the dental surgery 86
- — — effect of on tissue oxidation 24
- — — first use of as anaesthetic 6
- — — indications for 80
- — — in patients with cardiac failure 120
- — — induction of anaesthesia with 173-174
- — — intravenous administration of 10
- — — liquid vapour pressure of during administration of 348
- — — — — with Oxford Vaporiser 349-351
- — — physical properties of 173
- — — side actions of 17
- — — signs of too rapid administration of 174

- Ether use of in anaesthetic resistant patient 117
- Ethyl chloride 152-164
- — — administration of by closed method 161
- — — — — by open method 158
- — — advantages of 153
- — — as supplement to  $N_2O$  anaesthesia 162 213
- — — dangers of anaesthesia with 154-157
- — — disadvantages of 153
- — — first use of as anaesthetic 152
- — — in anaesthesia in children 127-129 153-162
- — — — — for patients with cleft palate 123
- — — indications for 153
- — — — — in dental surgery 86
- — — overdosage with 154-156
- — — physical properties of 152
- — — repeated applications of 157
- — — signs of anaesthesia with 154
- — — use of in anaesthetic resistant patient 117
- — — — — in insane patients 122
- Ethylene risk of explosion with 85
- Evipan 11 138 (*See also* Hexobarbitone)
- Lye examination of during anaesthesia 72-73
- — — signs of nitrous oxide anaesthesia 71-73

## F

- Fasting as cause of post anaesthetic nausea 243
- Fear reaction to anaesthesia 107-113
- Fergusson's mouth gag 271
- Flaxedil 198-199
- — — tachycardia due to 193
- Flowmeters 177
- Forceps tongue 282-284
- Foreign bodies in air passages 297 308-309
- — — — — impacted 313
- — — — — legal aspects of 358
- — — — — predisposing causes of 358-359
- — — — — removal of 314 324
- — — in alimentary tract 307
- — — in the mouth danger of during anaesthesia 306
- — — location of by X rays 360-362

## G

- Gags 271-276 (*See also* Mouth gags)
- Gallamine 198 (*See also* Flaxedil)
- Gangrene following intra arterial injection of thiopentone 148
- Gasdenal 137
- Gas inert effect of on tissue oxidation 24
- Gases anaesthetic respiratory exchange of 36-37
- — — Dalton's law 32

Gases estimation of amount of in cylinders 211 212  
 — exchange of in lungs 32-37  
 — transport of by blood 37-46  
 General anaesthesia in dentistry indications for 76-77  
 Glottis œdema of causing respiratory obstruction 301-304  
 Goldman's inhaler 166  
 Gourd curare 187

## H

Hæmoglobin content of blood 34  
 — in cyanosis 54-61  
 — oxygen association curve of 34  
 Head positioning of during extraction of teeth 233-239  
 Heart failure anaesthesia in presence of 120  
 — — during anaesthesia 332  
 — — — treatment of 332-333  
 — massage in treatment of heart failure 333  
 Hedonal intravenous 10  
 Hering Breuer reflex 50  
 Hewer's cardiac puncture needle 333  
 Hewitt's prop 277  
 Hexobarbitone 138  
 — as supplement to nitrous oxide 213  
 — discovery of 11  
 — indications for in dental surgery 86  
 — intravenous administration of 104  
 — rectal administration of 103  
 — use of in anaesthetic resistant patient 115 116  
 Histamine production of by *d* tubo curarine chloride 191 200  
 History of anaesthesia 1-12  
 Hospitalised patient preparation of for anaesthesia 135  
 Hydrogen ion concentration of aqueous solutions 39-41  
 Hyoscine combination of with morphia as premedicant 97  
 — in pre anaesthetic medication 96 105  
 Hyperpietic patient anaesthesia in 121  
 Hypnotics 137  
 Hypodermic syringe introduction of 2

## I

Inflator Oxford 327-329  
 Inhalation anaesthesia early experiments on 2-10  
 Inhaler Goldman's 166  
 — Loosely's 161  
 — Oxford 167 168  
 — T Young Simpson's 346  
 Insamity anaesthesia in 122  
 Insufflation method of anaesthesia 10  
 Intercostal muscles action of in respiration 30  
 Intestinal obstruction acute thiopentone contra indicated in 144

Intocostin 187 189  
 Intolerance to anaesthetics 112  
 Intratracheal anaesthesia 13  
 Intravenous administration of basal anaesthetics 104  
 — anaesthesia in dental surgery 86  
 Intubation nasal blind 246 260 265  
 — — direct vision 249-260  
 — under local anaesthesia 265

## J

Jaundice thiopentone contra indicated in 144  
 Jaw clenched method of opening of 272-274  
 — fracture of during dental anaesthesia 362-363  
 — — endotracheal anaesthesia in 266  
 Junker's bottle use of to produce analgesia 345-346

## K

Kirstein's autoscope 9

## L

Laborde's method of tongue traction 313  
 Laryngeal spasm 298-301 310-313  
 — — as cause of respiratory obstruction 298-301 310-313  
 — — causes of 310-313  
 — — in ether anaesthesia 174  
 — — in ethyl chloride anaesthesia 155  
 — — treatment of 312-313  
 Laryngoscope use of in direct vision intubation 249-260  
 Laryngoscopes 267-270  
 Laryngoscopy mechanics of 247-249  
 — under local anaesthesia 265-266  
 Laryngotomy 325-326  
 — tube 326  
 Larynx foreign bodies in 308-309 313-315  
 — intubation of for administration of anaesthetics 9 246  
 — occlusion of by spasm as cause of respiratory obstruction 298-301 310-313  
 Legal aspects of anaesthesia 356-365  
 — defence organisations 365  
 Local anaesthesia 13  
 — — in combination with regional anaesthesia 13  
 — — in dentistry indications for 77-78  
 Loosely's inhaler 161  
 Luminal 137  
 Lungs breathing area of 33  
 — capacity of 31  
 — diseases of anaesthesia in presence of 119  
 — fibrosis of as cause of anoxia 51  
 — gaseous exchange in 32-37  
 — toilet intubation for under local anaesthesia 266

## M

- Macbeth Bannister laryngoscope 269  
 Macintosh's bag 213-215  
 — laryngoscope 268  
 — mixing chamber 215  
 McKesson reducing valve 336  
 Mandrake soporific properties of 1  
 Mask Schummelbusch 158 175 182  
 Mason's mouth gag 271 364-365  
 Medication pre-anæsthetic 18 88-106  
 — atropine in 105 173 191  
 — basal anæsthetics in 99  
 — for ambulatory patient 93-95  
 — for hospitalised patient 95-106  
 — psychological 90  
 — sedatives used in 91-93  
 Medinal 137  
 Menstruation anæsthesia during 118  
 Mental attitude of patient towards anæsthesia 81-83  
 Midwifery analgesia self administered in 342-346  
 Minnitt gas and air apparatus reducing valves of 336-337  
 — — — use of to produce analgesia 343  
 Misconduct avoidance of charges of 242-243 364  
 Moir's apparatus use of to produce analgesia 344  
 Morphia addicts anæsthesia in 118  
 — as premedicant in ether anæsthesia 174  
 — combination of with hyoscine as premedicant 97  
 — depressant effect of on respiration 47  
 — in pre-anæsthetic medication 91-92  
 — — — of asthmatics 120  
 — — — of epileptics 122  
 — — — of hospitalised patient 95 96  
 — premedication in presence of pulmonary disease 119-120  
 — subcutaneous administration of 104  
 Morphine isolation of 2  
 Mouth foreign bodies in dangers of during anæsthesia 306  
 Mouth breathing during nasal nitrous oxide anæsthesia 225-230  
 Mouth-cover Paterson's 219  
 Mouth gags 271-276  
 — use of to open clenched jaw 272 274  
 Mouth-opener 276  
 Mouth packs description and use of 281-282  
 — as cause of respiratory obstruction 297 313-315  
 Mouth props description and use of 276-31  
 — too large as cause of respiratory obstruction 296  
 Muscles intercostal paralysis of as sign of anæsthesia 8 66  
 Muscle relaxants 186-203  
 Muscles respiratory 29-31

- Muscular signs of nitrous oxide anæsthesia 71  
 Mushin's prop 278  
 Myanesis 201-202  
 — in treatment of tetanus 201-202  
 — use of in anæsthesia 201-202

## N

- Narcotics first administration of in known doses 2  
 Nasal administration of nitrous oxide 222  
 — intubation blind 246 260-265  
 — direct vision 249-260  
 — under local anæsthesia 265-266  
 — respiration in dental anæsthesia 70 225-230  
 Nausea following nitrous-oxide administration 17 243-245  
 — post-anæsthetic 243  
 — treatment of 244  
 Nembutal 104 138  
 — as basal anæsthetic for children 125 126  
 — intravenous administration of 105  
 — oral administration of 104  
 Nepenthes narcotic effects of 1  
 Nervous patient anæsthesia in 107-112  
 Neuromuscular block 186-192  
 Nitrogen effect of on tissue oxidation 24  
 Nitrous oxide 211-245  
 — administration of errors in 218  
 — — in subjects with cardiac failure 120  
 — — in subjects with cleft palate 123  
 — — nasal 222  
 — — maintenance of anæsthesia in 230-234  
 — — prevention of discomfort during 219-221  
 — — advantages of in dental surgery 85  
 — — anæsthesia ethyl chloride as supplement to 162  
 — — hexobarbitone as supplement to 213  
 — — thiopentone as supplement to 140-143 213  
 — — premedication for 94  
 — — recovery from 240-245  
 — — resistance to 113-118  
 — — signs of 68-74  
 — — anæsthetic properties of discovery of 4-7  
 — — analgesic properties of discovery of 2-3  
 — — and air 231  
 — — and ether anæsthesia 9 177-178  
 — — and oxygen 231  
 — — asphyxia during administration of 69 218 310-312  
 — — asphyxia during administration of as cause of laryngeal spasm 310-312

- Nitrous oxide contra indicated in myocardial degeneration 25 120  
 — cyanosis during administration of 54-61  
 — Davy's experiments with 2-3  
 — effect of on tissue oxidation 25  
 — for analgesia 339-344  
 — general considerations 212-213  
 — in anaesthesia for children 126 127  
 — indications for use of 80  
 — in dental surgery 85  
 — in subjects with pulmonary disease 119  
 — intolerance to 112  
 — limitations of 212  
 — physical properties of 211  
 — potency of 27  
 — recovery from 240 245  
 — side actions of 17  
 — supplemented by ethyl chloride 162 213  
 — by hexobarbitone 213  
 — by thiopentone 140-143 213  
 — transport of in blood 39  
 Nose piece incorrect adjustment of as cause of respiratory obstruction 221 296

## O

- Obstetrics analgesia self administered in 342-346  
 Obstruction respiratory 291-304  
 — acute 310-315  
 — signs of 310  
 — causes of 291-304  
 — danger of in ethyl chloride anaesthesia 155  
 Oedema of glottis causing respiratory obstruction 301-304  
 Operation preparation of patient for 134-136  
 Opium use of as anodyne 2  
 — narcotic 2  
 Oral administration of basal anaesthetics 103  
 Overdosage of anaesthetic counter measures in 17 327-333  
 Oxford Inflator 327-328  
 — Inhaler 167 168  
 — Vaporiser 347-355  
 — methods of induction when using 353-355  
 — use of hand bellows 351-353  
 Oxidation tissue effect of anaesthetics on 24-25  
 Oxygen administration of during nitrous oxide anaesthesia 231-232  
 — and carbon dioxide exchange 34-36  
 — deprivation theory of anaesthesia 19-28  
 — inflation with as means of artificial respiration 327-329  
 — inhalation of therapeutic 2

- Oxygen tension (or pressure) of in alveolar air 34-36  
 — in cyanosis 56  
 — in blood reduction of effect on respiration 46  
 — transport of in blood 37  
 — utilisation of by body cells 20  
 Oxygen association curve of haemoglobin 38  
 Oxygen lack as cause of anaesthesia 19-28  
 — effect of on respiration 46-48  
 — on the sensitivity of the respiratory centre 48  
 — in nitrous-oxide anaesthesia 69-74 212 218 298

## P

- Pain relief of with opiates 2  
 Palate cleft anaesthesia in patients with 123  
 Pallor significance of during administration of ethyl chloride 156  
 Paraldehyde as basal anaesthetic for children 125  
 — method of rectal administration of 102  
 Paterson's mouth-cover 219  
 Patient ambulatory pre anaesthetic medication for 93-95  
 — complexion of during nitrous oxide anaesthesia 55-61  
 — consent of to be anaesthetised 364  
 — examination of prior to anaesthesia 206 363-364  
 — health of as guide to choice of anaesthetic 80  
 — hospitalised pre anaesthetic medication for 95  
 — mental attitude of towards anaesthesia 81-83 107-113  
 — nervous anaesthesia in 107-113  
 — position of in dental chair 206-208  
 — on operating table 285 286  
 — preparation of for operation 134-136  
 — resistance of to anaesthetics 113-118  
 — temperament of as indication of type of anaesthesia to be used 75-77  
 — unconscious care of 285-290  
 — moving of 286  
 — position of in bed 288  
 — responsibility for 286 364-365  
 Pectoral muscles action of in respiration 30  
 Pentamethonium iodide 200  
 Pentothal 138-151 (*See also* Thiopentone)  
 Pernoxon 138  
 pH of aqueous solutions 39-41  
 Phenobarbitone 137  
 — use of for premedication 94 122 136  
 Phenyl ethyl barbiturate 137  
 Phthisis anaesthesia in 77 120  
 — as cause of anoxia 51

Physiological processes effect of anæsthetics on 21  
 Physostigmine 190  
 Picrotoxin in treatment of barbiturate poisoning 151  
 — as antidote to myanesis 202  
 Pneumonia as cause of anoxia 51  
 Poppy soporific properties of 1  
 Position of patient in dental chair 206-208

— — — on operating table 285

Pot curare 187

Pre anæsthetic medication 18 89-106

— — — atropine in 105 173 191

— — — basal anæsthetics in 99

— — — for ambulatory patient 93-95

— — — for hospitalised patient 95-99

— — — psychological 90

— — — sedatives used in 91-93

Pregnancy anæsthesia in 119

Premedication 88-106 (*See also* Pre anæsthetic medication)

Preparation of patient for operation 134-136

Props 276-281 (*See also* Mouth props)

Prostigmin 190-191 197-198

— action of 190-191

— as antidote to *d* tubocurarine chloride 190-191 197-198

— as antidote to flaxedil 199

— dosage of 197

— effects of 190-191 197

Protocurarine 187

Protocuridine 187

Protocurine 187

Psychological disturbances during anæsthesia 222

— factors in administration of anæsthetics 107 113

— premedication 90

Pulmonary disease anæsthesia in presence of 119

Pulse during nitrous oxide anæsthesia 74

Pupils during nitrous oxide anæsthesia 73 74

## R

Radiological examination advisability of in case of missing foreign body 360-362

Recovery from nitrous oxide anæsthesia 240-245

Rectal anæsthesia first employment of 10  
 Rectum administration of anæsthetics per 10 101

Reducing valves 335-338

Regional anæsthesia 13

— — — in combination with local anæsthesia 13

Relaxants 186-203

Residual air 31

Resistance to anæsthetics 5 88-89 113-118

Respiration artificial 327-332

— during nitrous oxide anæsthesia 70-71

— effect of anæsthetics on 16

— mechanism of 29

— muscles employed in 29

— nasal in dental anæsthesia 70 223-230

— physico-chemical principles of 39-46

— physiology of 29-53

— regulation of 46

— stimulation of 48-49

Respiratory centre depression of 47

— — — as cause of anoxia 51

— — — effect of nervous stimuli on 50

— — — effect of thiopentone on 27

— — — paralysis of in fourth stage of anæsthesia 66

— — — ensitivity of 46-50

— obstruction 291-304

— — — acute 310-315

— — — signs of 310

— — — causes of 291-304

— — — danger of in ethyl chloride anæsthesia 155

— signs of nitrous oxide anæsthesia 70

— tract obstruction of as cause of anoxia 51

— — — during anæsthesia prevention of 232

Restlessness post operative 290

## S

Schaefer's method of artificial respiration 329-331

Schummelbusch's mask 158 175 182

Scopolamine combination of with morphia as premedicant 97

— in pre anæsthetic medication 96

Sedatives action of in pre anæsthetic medication 92

— use of in pre anæsthetic medication 91-99

Sensations during recovery from anæsthesia 240-245

Sensitivity to anæsthetics 131

Sepsis chronic anæsthesia in presence of 121

Shock anæsthesia in presence of 121

— contra indications to use of thiopentone following 144

Silvester's method of artificial respiration 331-332

Simpson's Inhaler use of to produce analgesia 346

Sodium amytal 138

Soporifics used in ancient times 1

Spasm laryngeal in ether anæsthesia 174

— — — as cause of respiratory obstruction 298-301 310-313

— — — causes of 310-313

— — — in ethyl chloride anæsthesia 155

— — — treatment of 312-313

- Spinal anaesthesia 13
- Sponge as cause of respiratory obstruction 247 314
- use of as mouth pack 281
- Status lymphaticus and anaesthesia 130-133
- Status periculosus* 131
- Sterno-mastoid muscle action of in respiration 30
- Stimuli response to in determination of depth of anaesthesia 64-65
- Stomach distension of during use of *d* tubocurarine chloride 194 195
- Strap use of in anaesthetic resistant patient 115 208 210
- Stridor in ethyl chloride anaesthesia 155
- Struggling during anaesthesia 65
- Sulphonamides cyanosis following administration of 54
- Syringe hypodermic introduction of 2

## T

- Tachypnoea following trilene inhalation 171
- Teeth extraction of 235-239
  - — — depth of anaesthesia necessary for 67
  - — — positioning of head for 235 231
  - — — under anaesthetics early experiments on 5-7
  - protection of during use of laryngoscope 251 362
- Tetanus treatment of 196 201
- Theory of anaesthesia 19-24
- Therapeutic quotient of anaesthetic 17
- Thio-barbiturates 138
- Thiopentone soluble 138 151
  - advantages of 138
  - as basal anaesthetic 103 105 139-142
  - as general anaesthetic 139-140
  - as supplement to nitrous oxide 140 143 213
  - contra indications to in dental surgery 66 143
  - dangers attending injection of into artery 148
  - dosage of 149-150
  - effect of on the respiratory centre 27
  - in anaesthesia for insane patients 122
  - — — of claustrophobic patient 122
  - — — of subjects with cleft palate 123
  - in war surgery 143
  - indications for use of in dental surgery 142
  - intra arterial injection avoidance of 149
  - intravenous administration of 104
  - overdosage with treatment of 151
  - rate of excretion of 150
  - rate of injection of 149
  - rectal administration of 103
  - signs of anaesthesia with 150
  - solution preparation of 138 139
- Thiopentone technique of administration of 145-150
  - use of in anaesthetic resistant patient 115-116 141 142
- Throat operations thiopentone contra indicated as sole anaesthetic in 144
- Tidal air 31
- Tissue oxidation effect of ether on 24
  - — — — inert gas on 24
  - — — — nitrous oxide on 25
- Tissues poisoning of as cause of anoxia 52
- Tobacco addicts anaesthesia in 118
- Tongue backward displacement of as cause of respiratory obstruction 291-296
  - traction Laborde's method 313
- Tongue forceps 242-244
- Trachea anatomy of 317
  - intubation of 246-267
- Tracheal tubes history of employment of in anaesthesia 9 10
- Tracheotomy after-care in 325
  - dangers of 324
  - emergency 315-325
  - operation for 318 324
  - instruments for 315 316
  - tube introduction of 323
- Trapezius muscles action of in respiration 30
- Trewby's apparatus 216 220 227
  - props 277
- Tribromethylalcohol 102
- Trichlorethylene 170-172 (*See also* Trilene)
- Trilene 170-172
  - analgesic value of 170
  - dangers of 171
  - first use of as anaesthetic 170
  - indications for use of 170
  - mode of administration of 171
  - physical properties of 170
  - production of dichloroacetylene from 171
  - toxic effects of 171
- Trismus use of mouth gag in 276
- Tubarine 188 (*See also d* tubocurarine chloride)
- Tubarine miscible 188
- Tube curare 187
- Tuberculous patient anaesthesia in 77 120
- Tubocurarine 187-198 (*See also d* tubocurarine chloride)

## U

- Unconsciousness determination of depth of 62-66

## V

- Valves cylinder 334-335
  - reducing 335-338
- Vaporiser Oxford 347-355
- Ventricular fibrillation 181







- Veronal 137  
 Vinesthene 165-169  
   — administration of by closed method 166  
   — — — by open method 168  
   — dosage of 166 168  
   — in dental surgery 165-169  
   — indications for in children 128  
   — — — in dental surgery 87  
   — physical properties of 165  
   — signs of anæsthesia with 166  
   — use of in anæsthetic resistant patient 117  
 Vinyl ether 165 169 (*See also* Vinesthene)  
 Vital capacity 31  
 Vocal cords adduction of in laryngeal spasm 298-301  
   — — difficulty of exposure of during intubation 257-265  
 Vomiting during anæsthesia for dental surgery 129 233  
   — — — during analgesia 342  
   — — — unconsciousness 289  
   — — — legal aspects of 365  
   — following nitrous oxide administration 17 243-245  
   — post anæsthetic 243-245  
 Vomitus as cause of respiratory obstruction 297
- W
- War surgery thiopentone in 143
- X
- X ray examination advisability of in cases of missing foreign body 360-362
- Y
- Young Simpson's inhaler use of to produce analgesia 346

